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=> s (NADPH(s)oxidase#) or (dual(s)oxidase#) or nox1 or noh1

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191 FILE WPINDEX
12 FILE IPA
13 FILE NAPRALERT
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L1 QUE (NADPH(S) OXIDASE#) OR (DUAL(S) OXIDASE#) OR NOX1 OR NOH1

=> d rank
F1 6596 BIOSIS
F2 6470 SCISEARCH
F3 6074 CAPLUS
F4 5483 TOXCENTER
F5 5207 MEDLINE
F6 3894 EMBASE
F7 2745 ESBIOBASE
F8 1910 PASCAL
F9 1744 BIOTECHNO
F10 1599 LIFESCI
F11 1365 USPATFULL
F12 845 CANCERLIT

=> file f1-f12
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CA INDEXING COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'CANCERLIT' ENTERED AT 10:04:59 ON 24 NOV 2005

=> s L1
L2 43932 L1

=> s (screen? or isolat? or find? or determ?) (s) L2
8 FILES SEARCHED...
L3 4878 (SCREEN? OR ISOLAT? OR FIND? OR DETERM?) (S) L2

=> s (screen? or isolat? or find? or determ? or identif?) (s) L2
7 FILES SEARCHED...
8 FILES SEARCHED...
L4 6284 (SCREEN? OR ISOLAT? OR FIND? OR DETERM? OR IDENTIF?) (S) L2

=> s (substanc? or compound? or inhibit? or antagoni?) (s) L4
8 FILES SEARCHED...
L5 2210 (SUBSTANC? OR COMPOUND? OR INHIBIT? OR ANTAGONI?) (S) L4

=> s (method? or process?) (s) L5
8 FILES SEARCHED...
L6 424 (METHOD? OR PROCESS?) (S) L5

=> s (gene# or sequence# or clone# or polynucleotide# or recombinant#) (s) L6
7 FILES SEARCHED...
9 FILES SEARCHED...
L7 93 (GENE# OR SEQUENCE# OR CLONE# OR POLYNUCLEOTIDE# OR RECOMBINANT#
) (S) L6

=> s disease# (s) L7
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=> s rheumatoid (s) L7
L9 1 RHEUMATOID (S) L7

=> dup rem l7
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L10 62 DUP REM L7 (31 DUPLICATES REMOVED)

=> d ibib abs l10 1-62

L10 ANSWER 1 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2005:219850 CAPLUS
DOCUMENT NUMBER: 142:292557
TITLE: Superoxide-generating oxidase Nox1 is functionally required for Ras oncogene transformation
INVENTOR(S): Mitsushita, Junji; Kamata, Tohru; Hirose, Kunitaka
PATENT ASSIGNEE(S): Kureha Chemical Industry Company, Limited, Japan
SOURCE: PCT Int. Appl., 76 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005021739	A1	20050310	WO 2004-JP11673	20040806

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 2003-308658 A 20030901
AB This invention relates to a polypeptide encoded by Nox1 gene, its homolog, a compn. for producing antibodies contg. Nox1 fragment, antibodies against Nox1, and a method of detecting mRNA expressing Nox1. The invention also provides a ***method*** of diagnosing caner with the use of ***Nox1*** ***gene*** assocd. with mutated Ras oncogene, a ***method*** of ***screening*** cancer growth ***inhibitors*** and medicinal compn. for use in treating cancer. PCR primers and probes targeting the Nox1 gene for cancer diagnosis and siRNA as anticancer agent are also provided. The activated Ras oncogene can transform various mammalian cells and has been implicated in development of a high population of malignant human tumors. Recent studies suggest that generation of reactive oxygen species such as superoxide and H2O2 is involved in cell transformation by the activated Ras. However, the nature of an oxidase participating in Ras-transformation is presently unknown.

Here, the authors report that Ras oncogene up-regulates the expression of Nox1, a homolog of the catalytic subunit of the superoxide-generating NADPH oxidase, via the mitogen-activated protein kinase kinase-mitogen-activated protein kinase pathway, and that small interfering RNAs designed to target Nox1 mRNA effectively blocks the Ras transformed phenotypes including anchorage-independent growth, morphol. changes, and prodn. of tumors in athymic mice. Therefore, they propose that increased reactive oxygen species generation by Ras-induced Nox1 is required for oncogenic Ras transformation.
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:292538 USPATFULL
TITLE: Use of a half-transporter protein of the abcg-family for selecting cells and in gene therapy
INVENTOR(S): Nemet, Katalin, Budapest, HUNGARY
Varady, Gyorgy, Budapest, HUNGARY
Cervenak, Judit, Budapest, HUNGARY
Ujhelly, Olga, Budapest, HUNGARY
Sarkadi, Balazs, Budapest, HUNGARY
Varadi, Andras, Budapest, HUNGARY
Ozvegy, Csilla, Budapest, HUNGARY
PATENT ASSIGNEE(S): SOLVO BIOTECHNOLOGY, Szeged, HUNGARY, H-6722 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005255084	A1	20051117
APPLICATION INFO.:	US 2003-493553	A1	20021024 (10)
	WO 2002-HU108		20021024
			20040423 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	HU 2003-P104446	20011024
	HU 2003-200015	20020304
	HU 2003-P203435	20021011

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WELSH & KATZ, LTD, 120 S RIVERSIDE PLAZA, 22ND FLOOR, CHICAGO, IL, 60606, US
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 1531
AB The invention relates to an isolated nucleic acid comprising a sequence encoding a half transporter protein of the ABCG-family for use in gene therapy, to the use of the isolated nucleic acid for selecting somatic mammalian cells against at least one drug transportable by the transporter protein, to vectors, cells, pharmaceutical compositions and kits comprising the nucleic acid and methods for protecting and selecting cells against a cytotoxic drug transportable by said transporter protein and for gene therapy methods.

L10 ANSWER 3 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:287473 USPATFULL
TITLE: Pharmaceutical dopamine glycoconjugate compositions and methods of their preparation and use
INVENTOR(S): christian, Samuel T., Chelsa, AL, UNITED STATES
Sundsmo, John S., Vista, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005250739	A1	20051110
APPLICATION INFO.:	US 2003-625645	A1	20030722 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-547501, filed on 12 Apr 2000, PENDING Continuation-in-part of Ser. No. US 2002-198798, filed on 18 Jul 2002, ABANDONED

Continuation-in-part of Ser. No. US 2000-547506, filed on 12 Apr 2000, GRANTED, Pat. No. US 6548484
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JOHN S. SUNDSMO, BIOMEDPATNET COM, P.O. BOX 535, VISTA, CA, 92085, US
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 3400
AB Hydrophilic transportable N-linked glycosyl dopaminergic prodrug compounds according to FORMULA V and methods of their use, ##STR1## wherein, Ring 1 comprises an aryl or heteroaryl ring having 4 to 8 carbon atoms, among which atoms are counted "X" and "Y";

each of X and Y is optional; X, when present is either --C(R.sub.1).sub.2-- or --C(R.sub.1).sub.2--; Y, when present, is either --CH.sub.2-- or --CH.sub.2--CH.sub.2--;
z, R.sub.5 and R.sub.5'. are optional, and when present z, R.sub.5 and R.sub.5' together form a lower alkyl or a substituted lower alkyl moiety; N is part of either an amine or an amide linkage; E is a saccharide which forms a linkage with N through a single bond from a carbon or oxygen atom thereof;
R.sub.1 and R.sub.4 are selected form the group consisting of hydrogen, hydroxyl, halogen, halo-lower alkyl, alkoxyl, alkoxyl-lower alkyl, halo-alkoxy, thioamido, amidosulfonyl, alkoxylcarbonyl, carboxamide, aminocarbonyl, and alkylamino-carbonyl;
R.sub.2 and R.sub.3 are hydroxyl;
R.sub.5 and R.sub.6, when present, are selected from the group consisting of hydrogen, hydroxyl, alkoxyl, carbonyl, alkoxylcarbonyl, aminocarbonyl, alkylamino-carbonyl and dialkylamino-carbonyl; and,
R.sub.6 and R.sub.6' are selected from the group consisting of hydrogen, hydroxyl, alkoxyl, carboxyl, alkoxylcarbonyl, aminocarbonyl, alkylamino-carbonyl and dialylamino-carbonyl, with the proviso that Ring 1 is capable of binding to any of: a dopaminergic receptor selected from the group consisting of a D1 receptor and a D5 receptor; a DAT transporter; a VMAT transporter; and, with the proviso that E is capable of binding to a GLUT transporter selected from the group consisting of a GLUT1 receptor and a GLUT3 receptor.

L10 ANSWER 4 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:248416 USPATFULL
TITLE: Medical compositions for intravesical treatment of bladder cancer
INVENTOR(S): Nuijen, Bastiaan, Amsterdam, NETHERLANDS
Pfadenhauer, Ernie, Irvine, CA, UNITED STATES
Beijnen, Jos H., Amsterdam, NETHERLANDS
PATENT ASSIGNEE(S): Spectrum Pharmaceuticals, Inc., Irvine, CA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005215615	A1	20050929
APPLICATION INFO.:	US 2005-96566	A1	20050401 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2002-285783, filed on 1 Nov 2002, GRANTED, Pat. No. US 6894071

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-344446P	20011101 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PRESTON GATES & ELLIS LLP, 1900 MAIN STREET, SUITE 600, IRVINE, CA, 92614-7319, US
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
LINE COUNT: 974
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Anti-cancer coating compositions comprising 3-hydroxymethyl-5-aziridinyl-

1-1-methyl-2-[1 H-indole-4,7-dione]propenol (E09) are disclosed. More specifically, the coating compositions comprise EO9 and a formulation vehicle. The formulation vehicle improves the solubility and stability of EO9. Additionally, the coating compositions can include coating agents that provide better adhesion of the coating composition to the bladder wall during intravesical delivery of the coating composition.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 5 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:241682 USPATFULL
TITLE: Artificial vessel scaffold and artifical organs therefrom
INVENTOR(S): Sitzmann, James V., Potomac, MD, UNITED STATES
Sitzmann, Eugene V., Cooke, IL, UNITED STATES
PATENT ASSIGNEE(S): Bioartis, Inc., Pittsford, NY, UNITED STATES, 14534 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005209687	A1	20050922
APPLICATION INFO.:	US 2003-505131	A1	20030219 (10)
	WO 2003-US4505		20030219
			20050420 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-357118P	20020219 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CROWELL & MORING LLP, INTELLECTUAL PROPERTY GROUP, P.O. BOX 14300, WASHINGTON, DC, 20044-4300, US
NUMBER OF CLAIMS: 39
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 2142
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An artificial vessel scaffold is provided, of biocompatible materials and capable of being coated with selected cell types. A plurality of artificial organs are provided, formed of a biocompatible scaffold material and coated with selected cell types.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 6 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:234351 USPATFULL
TITLE: Nanoparticulate probe for in vivo monitoring of tissue oxygenation
INVENTOR(S): Kuppusamy, Periannan, New Albany, OH, UNITED STATES
Pandian, Ramasamy P., Columbus, OH, UNITED STATES
Parinandi, Narasimham L., Upper Arlington, OH, UNITED STATES
Zweier, Jay L., Blacklick, OH, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005203292	A1	20050915
APPLICATION INFO.:	US 2004-935297	A1	20040907 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-500714P	20030905 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CALFEE HALTER & GRISWOLD, LLP, 800 SUPERIOR AVENUE, SUITE 1400, CLEVELAND, OH, 44114, US
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 2922

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A new class of micro- and nano-particulate paramagnetic spin probes especially useful for magnetic resonance imaging techinques, including electron paramagnetic resonance (EPR) and magnetic resonance imaging (MRI). The probes are lithium phthalocyanine derivative compounds. Also provided are are suspensions and emulsions comprising lithium phthalocyanine derivative probes. Also provided are noninvasive methods for measuring noninvasive methods of measuring oxygen concentration, oxygen partial pressure, oxygen metabolism, and nitric oxide concentration in a specific tissue, organ, or cell in vivo or in vitro.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 7 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:226938 USPATFULL
TITLE: Methods and compositions for NAD(P)(H) oxidases
INVENTOR(S): Renate Else Bommarius, Bettina, Atlanta, GA, UNITED STATES
Bommarius, Andreas Sebastian, Atlanta, GA, UNITED STATES
Gibbs, Phillip Ray, Atlanta, GA, UNITED STATES
Wellborn, William Benjamin, Marietta, GA, UNITED STATES
PATENT ASSIGNEE(S): Georgia Tech Research Corporation, Atlanta, GA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005196788	A1	20050908
APPLICATION INFO.:	US 2005-45874	A1	20050128 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 2003-US24067, filed on 31 Jul 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-399850P	20020731 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TROUTMAN SANDERS LLP, BANK OF AMERICA PLAZA, SUITE 5200, 600 PEACHTREE STREET , NE, ATLANTA, GA, 30308-2216, US
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1-51
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 2898
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention is directed to compositions and methods comprising NAD(P)H oxidases, particularly bacterial oxidases, nucleic acids, recombinant plasmid vectors and recombinant proteins therein encoded, and host cells comprising the oxidases and nucleic acids. The present invention also comprises an isolated bacterial oxidase that oxidizes both NADH and NADPH. Methods for producing the enzymes and enzymatic reactions comprising use of NAD(P)H oxidases and products of such reactions are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 8 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:203201 USPATFULL
TITLE: Genes differentially expressed by acutely isolated resident progenitor cells of the human white matter
INVENTOR(S): Goldman, Steven A., Webster, NY, UNITED STATES
Sim, Fraser, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005176626	A1	20050811
APPLICATION INFO.:	US 2004-985306	A1	20041110 (10)

	NUMBER	DATE

PRIORITY INFORMATION: US 2003-519310P 20031110 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Michael L. Goldman, Nixon Peabody LLP, Clinton Square, P.O. Box 31051, Rochester, NY, 14603-1051, US
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 2135
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a method of modulating production of neurons and/or oligodendrocytes from neural progenitor cells of human white matter and to a method of treating a subject for a condition modulated by underproduction of oligodendrocytes from human white matter. Both of these methods involve administering an agonist or antagonist of one or more molecules set forth in Tables 1 and/or 2 to the neural progenitor cells. Also disclosed is a method of using an inhibitor of sterol synthesis to differentiate oligodendrocyte progenitor cells to oligodendrocytes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 9 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:197007 USPATFULL
TITLE: Methods of lowering lipid levels in a mammal
INVENTOR(S): Rahbar, Samuel, Beverly Hills, CA, UNITED STATES
Figarola, James L., Hacienda Heights, CA, UNITED STATES
PATENT ASSIGNEE(S): City of Hope, Duarte, CA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005171150	A1	20050804
APPLICATION INFO.:	US 2004-974028	A1	20041027 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-514476P	20031027 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ROTHWELL, FIGG, ERNST & MANBECK, P.C., 1425 K STREET, N.W., SUITE 800, WASHINGTON, DC, 20005, US
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 23 Drawing Page(s)
LINE COUNT: 1719
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to methods for lowering lipid levels in mammals using compounds that inhibit advanced glycation endproducts (AGEs), LR-9, LR-74 and LR-90. These compounds, which inhibit non-enzymatic protein glycation, also inhibit the formation of advanced lipoxidation endproducts (ALEs) on target proteins by trapping intermediates in glycoxidation and lopoxidation and inhibiting oxidation reactions important in the formation of AGEs and ALEs.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 10 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:171886 USPATFULL
TITLE: 3-imino-2-indolones for the treatment of depression and/or anxiety
INVENTOR(S): Konkel, Michael, Garfield, NJ, UNITED STATES
Wetzel, John M., Fairlawn, NJ, UNITED STATES
Talisman, Jamie, New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005148635	A1	20050707
APPLICATION INFO.:	US 2005-68203	A1	20050228 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2003-637971, filed on 7 Aug

2003, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-402025P	20020807 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LUNDBECK RESEARCH USA, INC., ATTENTION: STEPHEN G. KALINCHAK, LEGAL, 215 COLLEGE ROAD, PARAMUS, NJ, 07652, US
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1-8
LINE COUNT: 1666
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention is directed to indolone derivatives which are selective antagonists for the GalR3 receptor. The invention provides a pharmaceutical composition comprising a therapeutically effective amount of a compound of the invention and a pharmaceutically acceptable carrier. This invention also provides a pharmaceutical composition made by combining a therapeutically effective amount of a compound of the invention and a pharmaceutically acceptable carrier. This invention further provides a process for making a pharmaceutical composition comprising combining a therapeutically effective amount of a compound of the invention and a pharmaceutically acceptable carrier. This invention also provides a method of treating a subject suffering from depression and/or anxiety which comprises administering to the subject an amount of a compound of the invention effective to treat the subject's depression and/or anxiety. This invention also provides a method of treating depression and/or anxiety in a subject which comprises administering to the subject a composition comprising a pharmaceutically acceptable carrier and a therapeutically effective amount of a GalR3 receptor antagonist.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 11 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:158186 USPATFULL
TITLE: Cell-based assay for identifying peptidase inhibitors
INVENTOR(S): Fang, Hong, Chapmansboro, TN, UNITED STATES
Green, Neil, Chapmansboro, TN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005136394	A1	20050623
APPLICATION INFO.:	US 2004-842846	A1	20040511 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-480625P	20030623 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FULBRIGHT & JAWORSKI L.L.P., SUITE 2400, 600 CONGRESS AVENUE, AUSTIN, TX, 78701-3271, US
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 2115
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides assays for the identification of inhibitors of endopeptidase toxins. The assays utilize genetically engineered yeast cells that contain a conditionally expressed endopeptidase toxin. When conditions for expression of the toxin are met, the toxin cleaves a yeast (natural or engineered) peptide product that is required for yeast survival. If the yeast is grown in the presence of an candidate substance that is an inhibitor of the toxin, the yeast survives, thereby providing a rapid and sensitive identification of the inhibitor.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 12 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:144321 USPATFULL
TITLE: Novel oxidase
INVENTOR(S): Kawakami, Masakatsu, Tsukuba-shi, JAPAN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005124056	A1	20050609
APPLICATION INFO.:	US 2003-509622	A1	20030605 (10)
	WO 2003-JP7148		20030605

	NUMBER	DATE
PRIORITY INFORMATION:	JP 2003-2002165612	20020606
	JP 2003-60749	20030307

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SUGHRUE MION, PLLC, 2100 PENNSYLVANIA AVENUE, N.W., SUITE 800, WASHINGTON, DC, 20037, US
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 1230
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB There is disclosed an oxidase gene useful for the diagnosis of RA and the screening of a substance for the treatment of RA and/or a substance for the treatment of osteoarthritis. Also, an inspection method useful as a diagnosis method for RA is disclosed. Additionally, there is disclosed a method for screening a substance for the treatment of RA and/or a substance for the treatment of osteoarthritis, using the aforementioned novel oxidase gene. Also disclosed is a method for producing a pharmaceutical composition for the treatment of RA and/or the treatment of osteoarthritis which comprises an inhibitor of the aforementioned oxidase, which is obtainable by the aforementioned screening method, as an active ingredient.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 13 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:144171 USPATFULL
TITLE: Protein modulation
INVENTOR(S): Rana, Tariq M., Shrewsbury, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005123906	A1	20050609
APPLICATION INFO.:	US 2004-984946	A1	20041108 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-518543P	20031106 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110, US
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 1180
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods for screening and identifying compounds that inhibit a pathway affecting protein levels, and methods and compounds for treating viral, e.g., HIV, infection.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 14 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:139784 USPATFULL
TITLE: Inbred corn line PHADA
INVENTOR(S): Benson, David Lee, York, NE, UNITED STATES

PATENT ASSIGNEE(S): Pioneer Hi-Bred International, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005120439	A1	20050602
APPLICATION INFO.:	US 2005-48442	A1	20050131 (11)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PIONEER HI-BRED INTERNATIONAL INC., 7100 N.W. 62ND AVENUE, P.O. BOX 1000, JOHNSTON, IA, 50131, US
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
LINE COUNT: 3112
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A novel inbred maize line designated PHADA and seed, plants and plant parts thereof. Methods for producing a maize plant that comprise crossing inbred maize line PHADA with another maize plant. Methods for producing a maize plant containing in its genetic material one or more traits introgressed into PHADA through backcross conversion and/or transformation, and to the maize seed, plant and plant part produced thereby. Hybrid maize seed, plant or plant part produced by crossing the inbred line PHADA or a trait conversion of PHADA with another maize line. Inbred maize lines derived from inbred maize line PHADA, methods for producing other inbred maize lines derived from inbred maize line PHADA and the inbred maize lines and their parts derived by the use of those methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 15 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:139783 USPATFULL
TITLE: Hybrid maize 37F73
INVENTOR(S): Kevern, Thomas Craig, Milton, WI, UNITED STATES
PATENT ASSIGNEE(S): Pioneer Hi-Bred International, Inc., Johnston, IA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005120438	A1	20050602
APPLICATION INFO.:	US 2005-48371	A1	20050131 (11)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCKEE, VOORHEES & SEASE, P.L.C., ATTN: PIONEER HI-BRED, 801 GRAND AVENUE, SUITE 3200, DES MOINES, IA, 50309-2721, US
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
LINE COUNT: 2753
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A novel hybrid maize variety designated 37F73 and seed, plants and plant parts thereof, produced by crossing two Pioneer Hi-Bred International, Inc. proprietary inbred maize lines. Methods for producing a maize plant that comprises crossing hybrid maize variety 37F73 with another maize plant. Methods for producing a maize plant containing in its genetic material one or more traits introgressed into 37F73 through backcross conversion and/or transformation, and to the maize seed, plant and plant part produced thereby. This invention relates to the hybrid seed 37F73, the hybrid plant produced from the seed, and variants, mutants, and trivial modifications of hybrid 37F73. This invention further relates to methods for producing maize lines derived from hybrid maize variety 37F73 and to the maize lines derived by the use of those methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 16 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:139780 USPATFULL
TITLE: Soybean variety XB25C05
INVENTOR(S): Streit, Leon George, Johnston, IA, UNITED STATES
Stephens, Paul Alan, Princeton, IL, UNITED STATES
PATENT ASSIGNEE(S): Pioneer Hi-Bred International, Inc. (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2005120435 A1 20050602
APPLICATION INFO.: US 2005-48688 A1 20050131 (11)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PIONEER HI-BRED INTERNATIONAL INC., 7100 N.W. 62ND AVENUE, P.O. BOX 1000, JOHNSTON, IA, 50131, US
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
LINE COUNT: 1693
AB According to the invention, there is provided a novel soybean variety designated XB25C05. This invention thus relates to the seeds of soybean variety XB25C05, to the plants of soybean XB25C05 to plant parts of soybean variety XB25C05 and to methods for producing a soybean plant produced by crossing plants of the soybean variety XB25C05 with another soybean plant, using XB25C05 as either the male or the female parent.

L10 ANSWER 17 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:139779 USPATFULL
TITLE: Soybean variety 90M01
INVENTOR(S): Roach, Michael Thomas, Redwood Falls, MN, UNITED STATES
Fabrizius, Martin Arthur, Redwood Falls, MN, UNITED STATES
PATENT ASSIGNEE(S): Pioneer Hi-Bred International, Inc. (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2005120434 A1 20050602
APPLICATION INFO.: US 2005-48535 A1 20050131 (11)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PIONEER HI-BRED INTERNATIONAL INC., 7100 N.W. 62ND AVENUE, P.O. BOX 1000, JOHNSTON, IA, 50131, US
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
LINE COUNT: 1688
AB According to the invention, there is provided a novel soybean variety designated 90M01. This invention thus relates to the seeds of soybean variety 90M01, to the plants of soybean 90M01 to plant parts of soybean variety 90M01 and to methods for producing a soybean plant produced by crossing plants of the soybean variety 90M01 with another soybean plant, using 90M01 as either the male or the female parent.

L10 ANSWER 18 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:139772 USPATFULL
TITLE: Soybean variety XB43D05
INVENTOR(S): Thompson, Jeffrey Allan, Edwardsville, IL, UNITED STATES
Streit, Leon George, Johnston, IA, UNITED STATES
PATENT ASSIGNEE(S): Pioneer Hi-Bred International, Inc. (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2005120427 A1 20050602
APPLICATION INFO.: US 2005-48362 A1 20050131 (11)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PIONEER HI-BRED INTERNATIONAL INC., 7100 N.W. 62ND AVENUE, P.O. BOX 1000, JOHNSTON, IA, 50131, US
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
LINE COUNT: 1691
AB According to the invention, there is provided a novel soybean variety designated XB43D05. This invention thus relates to the seeds of soybean variety XB43D05, to the plants of soybean XB43D05 to plant parts of soybean variety XB43D05 and to methods for producing a soybean plant

produced by crossing plants of the soybean variety XB43D05 with another soybean plant, using XB43D05 as either the male or the female parent.

L10 ANSWER 19 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:139770 USPATFULL
TITLE: Soybean variety XB39N05
INVENTOR(S): Corbin, Thomas Charles, Monticello, IL, UNITED STATES
Streit, Leon George, Johnston, IA, UNITED STATES
PATENT ASSIGNEE(S): Pioneer Hi-Bred International, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005120425	A1	20050602
APPLICATION INFO.:	US 2005-48357	A1	20050131 (11)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PIONEER HI-BRED INTERNATIONAL INC., 7100 N.W. 62ND AVENUE, P.O. BOX 1000, JOHNSTON, IA, 50131, US
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
LINE COUNT: 1693
AB According to the invention, there is provided a novel soybean variety designated XB39N05. This invention thus relates to the seeds of soybean variety XB39N05, to the plants of soybean XB39N05 to plant parts of soybean variety XB39N05 and to methods for producing a soybean plant produced by crossing plants of the soybean variety XB39N05 with another soybean plant, using XB39N05 as either the male or the female parent.

L10 ANSWER 20 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:137957 USPATFULL
TITLE: Target detection system having a conformationally sensitive probe comprising a nucleic acid based signal transducer
INVENTOR(S): Chun, Keun Ho, Seoul, KOREA, REPUBLIC OF
Hwang, Hyun Jin, Seoul, KOREA, REPUBLIC OF
PATENT ASSIGNEE(S): Ahram Biosystems Inc. (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005118603	A1	20050602
APPLICATION INFO.:	US 2003-684346	A1	20031010 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-684230, filed on 10 Oct 2003, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-417864P	20021011 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: EDWARDS & ANGELL, LLP, P.O. BOX 55874, BOSTON, MA, 02205, US
NUMBER OF CLAIMS: 228
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 52 Drawing Page(s)
LINE COUNT: 6599
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a system for detecting at least one target agent in a sample. The system generally includes at least one probe adapated to relate presence of the target agent to a detectable change in probe conformation. Preferred probes include a conformationally responsive signal transducer that reports association of the target agent and the probe by detectably shifting from one hybridization state to another. The invention has a wide spectrum of important applications including use in the rapid detection of target agents in biological, industrial, and environmental samples.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 21 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:112219 USPATFULL
TITLE: Method of treating cancer using dithiocarbamate derivatives
INVENTOR(S): White, David, Chicago, IL, UNITED STATES
Whittle, Robert R., Wilmington, NC, UNITED STATES
Stowell, Grayson Walker, Wilmington, NC, UNITED STATES
Whittall, Linda B., Wilmington, NC, UNITED STATES
Kennedy, Thomas, Charlotte, NC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005096304	A1	20050505
APPLICATION INFO.:	US 2004-922728	A1	20040820 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-378206, filed on 3 Mar 2003, PENDING Division of Ser. No. US 2000-735205, filed on 12 Dec 2000, GRANTED, Pat. No. US 6548540 Continuation-in-part of Ser. No. US 2000-679932, filed on 5 Oct 2000, GRANTED, Pat. No. US 6706759 Continuation-in-part of Ser. No. US 1999-392122, filed on 8 Sep 1999, GRANTED, Pat. No. US 6589987

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-99390P	19980908 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF LLP, 300 S. WACKER DRIVE, 32ND FLOOR, CHICAGO, IL, 60606, US
NUMBER OF CLAIMS: 62
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 4815
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention encompasses neutral dithiocarbamate metal compounds and methods of treating cancer using such compounds, along with methods for sensitizing AIDS/HIV patients to anti-retroviral therapy by blocking the P-glycoprotein membrane toxin extrusion pump using such compounds. Compounds inhibit the growth of cancer cells of a variety of cell types. A method is presented for using the neutral compounds disclosed herein, amongst other uses disclosed herein, to reduce tumor growth, and to potentiate the effect of other anticancer agents. The invention also encompasses pharmaceutical compositions comprising the neutral compounds and a pharmaceutically acceptable excipient, diluent, solubilizer, solvent, adjuvant or carrier, or a mixture thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 22 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:106763 USPATFULL
TITLE: Ant2 conditional knockout mouse and methods
INVENTOR(S): Wallace, Douglas C., Irvine, CA, UNITED STATES
MacGregor, Grant, Irvine, CA, UNITED STATES
Waymire, Katrina, Irvine, CA, UNITED STATES
Levy, Shawn E., Brentwood, TN, UNITED STATES
Sligh, James E., Brentwood, TN, UNITED STATES
Kokoszka, Jason E., Frederick, MD, UNITED STATES
PATENT ASSIGNEE(S): Emory University, Atlanta, GA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005091704	A1	20050428
APPLICATION INFO.:	US 2003-654628	A1	20030902 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-407364P	20020830 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GREENLEE WINNER AND SULLIVAN P C, 4875 PEARL EAST CIRCLE, SUITE 200, BOULDER, CO, 80301, US
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 1178
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Described are methods for inactivating adenine nucleotide transporter proteins in specific tissues of a transgenic nonhuman animal using a conditional knockin/knockout technology such as the Cre-LoxP, Flip-FLP recombinase, or Tet-on/off technologies. Specifically, the Ant2 gene is functionally inactivated in a mouse in liver, with or without the concurrent inactivation of the Ant1 gene. The result is an animal in which the Ant2 gene and accompanying ANT2 protein is absent in one or more tissues, either in the presence or absence of the Ant1 gene and accompanying ANT1 protein. The resulting animals, cells, mitochondria, and subcelluar fractions such as the mitochondrial permeability transition pore can then be used to identify agents that affect animal and/or subcellular function via a direct or indirect interaction with the ANT2 protein and/or its Ant2 gene.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 23 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:82639 USPATFULL
TITLE: Gene expression profile biomarkers and therapeutic targets for brain aging and age-related cognitive impairment
INVENTOR(S): Landfield, Philip W, Lexington, KY, UNITED STATES
Blalock, Eric M, Lexington, MA, UNITED STATES
Chan, Kuay-Chu, Lexington, KY, UNITED STATES
Fossex, Thomas, Gainesville, FL, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005071088	A1	20050331
APPLICATION INFO.:	US 2004-486706	A1	20040813 (10)
	WO 2002-US25607		20020813

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-311343P	20010813 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: McDermott Will & Emery, 600 13th Street NW, Washington, DC, 20005-3096
NUMBER OF CLAIMS: 69
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 3542
AB A statistical and functional correlation strategy to identify changes in cellular pathways specifically linked to impaired cognitive function with aging. Analyses using the strategy identified multiple groups of genes expressed in the hippocampi of mammals, where the genes were expressed at different levels for several ages. The aging changes in expression began before mid-life. Many of the genes were involved in specific neuronal and glial pathways with previously unrecognized relationships to aging and/or cognitive decline. The processes identified by the strategy suggest a new hypothesis of brain aging in which initially decreased neuronal activity and/or oxidative metabolism trigger separate but parallel genomic cascades in neurons and glia. In neurons, the cascade results in elevations in calcium signaling and reductions of immediate early gene signaling, biosynthesis, synaptogenesis and neurite remodeling. In contrast, glia undergo increased lipid metabolism and mediate a cycle of demyelination and remyelination that induces antigen presentation, inflammation, oxidative stress and extracellular restructuring.

These identified genes and the proteins they encode can be used as novel

biomarkers of brain aging and as targets for developing treatment methods against age-related cognitive decline, Alzheimer's Disease and Parkinson's Disease.

L10 ANSWER 24 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2005:217314 USPATFULL
TITLE: Attenuated salmonella SP12 mutants as antigen carriers
INVENTOR(S): Hensel, Michael, Munich, GERMANY, FEDERAL REPUBLIC OF
Holden, David William, London, UNITED KINGDOM
Shea, Jacqueline Elizabeth, High Wycombe, UNITED KINGDOM
PATENT ASSIGNEE(S): Microscience Limited, Wokingham Berkshire, UNITED KINGDOM (non-U.S. corporation)
Imperial College Innovations Limited, London, UNITED KINGDOM (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6936425	B1	20050830
	WO 2000014240		20000316
APPLICATION INFO.:	US 2001-763620		19990903 (9)
	WO 1999-EP6514		19990903
			20020301 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	EP 2001-98116827	19980904

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Swartz, Rodney P.
ASSISTANT EXAMINER: Shahnan-Shah, Khatol S
LEGAL REPRESENTATIVE: Holland & Knight LLP
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 69 Drawing Figure(s); 31 Drawing Page(s)
LINE COUNT: 5135
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to vaccines, in particular, to an attenuated gram-negative cell comprising the SP12 gene locus, wherein at least one gene of the SP12 locus is inactivated, wherein the inactivation results in an attenuation/reduction of virulence compared to the wild type of said cell, and to a carrier for the presentation of an antigen to a host, which carrier is the attenuated gram-negative cell, wherein the cell comprises at least one heterologous nucleic acid molecule comprising a nucleic acid sequence coding for the antigen, wherein the cell is capable of expressing the nucleic acid molecule or capable of causing the expression of the nucleic acid molecule in a target cell.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 25 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2005096975 ESBIOBASE
TITLE: Environmental pollutant and potent mutagen 3-nitrobenzanthrone forms DNA adducts after reduction by NAD(P)H:quinone oxidoreductase and conjugation by acetyltranferases and sulfotransferases in human hepatic cytosols
AUTHOR: Arlt V.M.; Stiborova M.; Henderson C.J.; Osborne M.R.; Bieler C.A.; Frei E.; Martinek V.; Sopko B.; Wolf C.R.; Schmeiser H.H.; Phillips D.H.
CORPORATE SOURCE: V.M. Arlt, Section of Molecular Carcinogenesis, Institute of Cancer Research, Brookes Lawley Building, Cotswold Road, Sutton, Surrey SM2 5NG, United Kingdom. E-mail: volker.arlt@icr.ac.uk
SOURCE: Cancer Research, (01 APR 2005), 65/7 (2644-2652), 51 reference(s)
CODEN: CNREA8 ISSN: 0008-5472

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB 3-Nitrobenzanthrone (3-nitro-7H-benz[de]anthracen-7-one, 3-NBA) is a potent mutagen and suspected human carcinogen ***identified*** in diesel exhaust and air pollution. We compared the ability of human hepatic cytosolic samples to catalyze DNA adduct formation by 3-NBA. Using the .sup.3.sup.2P-postlabeling ***method*** , we found that 12/12 hepatic cytosols activated 3-NBA to form multiple DNA adducts similar to those formed in vivo in rodents. By comparing 3-NBA-DNA adduct formation in the presence of cofactors of NAD(P)H:quinone oxidoreductase (NQO1) and xanthine ***oxidase*** , most of the reductive activation of 3-NBA in human hepatic cytosols was attributed to NQO1. ***Inhibition*** of adduct formation by dicoumarol, an NQO1 ***inhibitor*** , supported this ***finding*** and was confirmed with human ***recombinant*** NQO1. When cofactors of N,O-acelyltransferases (NAT) and sulfotransferases (SULT) were added to cytosolic samples, 3-NBA-DNA adduct formation increased 10- to 35-fold. Using human ***recombinant*** NQO1 and NATs or SULTs, we found that mainly NAT2, followed by SULT1A2, NAT1, and, to a lesser extent, SULT1A1 activate 3-NBA. We a so evaluated the role of hepatic ***NADPH*** :cytochrome P450 oxidoreductase (POR) in the activation of 3-NBA in vivo by treating hepatic POR-null mice and wild-type littermates i.p. with 0.2 or 2 mg/kg body weight of 3-NBA. No difference in DNA binding was found in any tissue examined (liver, lung, kidney, bladder, and colon) between null and wild-type mice, indicating that 3-NBA is predominantly activated by cytosolic nitroreductases rather than microsomal POR. Collectively, these results show the role of human hepatic NQO1 to reduce 3-NBA to species being further activated by NATs and SULTs. .COPYRGT.2005 American Association for Cancer Research.

L10 ANSWER 26 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2005253974 ESBIOBASE
TITLE: NADPH oxidase mediates hypersomnolence and brain oxidative injury in a murine model of sleep apnea
AUTHOR: Zhan G.; Serrano F.; Fenik P.; Hsu R.; Kong L.; Pratico D.; Klann E.; Veasey S.C.
CORPORATE SOURCE: Dr. S.C. Veasey, Center for Sleep and Respiratory Neurobiology, Department of Medicine, University of Pennsylvania School of Medicine, 3600 Spruce St., Philadelphia, PA 19104, United States.
E-mail: veasey@mail.med.upenn.edu
SOURCE: American Journal of Respiratory and Critical Care Medicine, (01 OCT 2005), 172/7 (921-929), 55 reference(s)
CODEN: AJCMED ISSN: 1073-449X
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Rationale: Persons with obstructive sleep apnea may have significant residual hypersomnolence, despite therapy. Long-term hypoxia/reoxygenation events in adult mice, simulating oxygenation patterns of moderate-severe sleep apnea, result in lasting hypersomnolence, oxidative injury, and proinflammatory responses in wake-active brain regions. We hypothesized that long-term intermittent hypoxia activates brain ***NADPH*** ***oxidase*** and that this enzyme serves as a critical source of superoxide in the oxidation injury and in hypersomnolence. Objectives: We sought to ***determine*** whether long-term hypoxia/reoxygenation events in mice result in ***NADPH*** ***oxidase*** activation and whether ***NADPH*** ***oxidase*** is essential for the proinflammatory response and hypersomnolence. ***Methods*** : ***NADPH*** ***oxidase*** ***gene*** and protein responses were measured in wake-active brain regions in wild-type mice exposed to long-term hypoxia/reoxygenation. Sleep and oxidative and proinflammatory responses were measured in adult mice either devoid of ***NADPH*** ***oxidase*** activity (gp91 .sup.p.sup.h.sup.o.sup.x-null mice) or in which ***NADPH***

oxidase activity was systemically ***inhibited*** with apocynin osmotic pumps throughout hypoxia/reoxygenation. Main Results: Long-term intermittent hypoxia increased ***NADPH*** ***oxidase*** ***gene*** and protein responses in wake-active brain regions. Both transgenic absence and pharmacologic ***inhibition*** of ***NADPH*** ***oxidase*** activity throughout long-term hypoxia/reoxygenation conferred resistance to not only long-term hypoxia/reoxygenation hypersomnolence but also to carbonylation, lipid peroxidation injury, and the proinflammatory response, including inducible nitric oxide synthase activity in wake-active brain regions. Conclusions: Collectively, these ***findings*** strongly support a critical role for ***NADPH*** ***oxidase*** in the lasting hypersomnolence and oxidative and proinflammatory responses after hypoxia/reoxygenation patterns simulating severe obstructive sleep apnea oxygenation, highlighting the potential of ***inhibiting*** ***NADPH*** ***oxidase*** to prevent oxidation-mediated morbidities in obstructive sleep apnea.

L10 ANSWER 27 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2005134553 ESBIOBASE
TITLE: Diphenyleneiodium (DPI) reduces oxalate ion- and calcium oxalate monohydrate and brushite crystal-induced upregulation of MCP-1 in NRK 52E cells
AUTHOR: Umekawa T.; Byer K.; Uemura H.; Khan S.R.
CORPORATE SOURCE: S.R. Khan, Department of Pathology, Laboratory Medicine, University of Florida College of Medicine, Box 100275, Gainesville, FL 32610-0275, Japan. E-mail: Khan@pathology.ufl.edu
SOURCE: Nephrology Dialysis Transplantation, (2005), 20/5 (870-878), 30 reference(s) CODEN: NDTREA ISSN: 0931-0509
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background. Our earlier studies have demonstrated upregulation of monocyte chemoattractant protein-1 (MCP-1) in NRK52F rat renal epithelial cells by exposure to oxalate (Ox) ions and crystals of calcium oxalate monohydrate (COM) or the brushite (Br) form of calcium phosphate. The upregulation was mediated by reactive oxygen species (ROS). This study was performed to investigate whether ***NADPH*** ***oxidase*** is involved in ROS production. ***Methods*** . Confluent cultures of NRK52E cells were exposed to Ox ions or COM and Br crystals. They were exposed for 1, 3, 6, 12, 24 and 48 h for ***isolation*** of MCP-1 mRNA and 24h for enzyme-linked immunosorbent assay (ELISA) to ***determine*** the secretion of protein into the culture medium. We also investigated the effect of free radical scavenger, catalase, and the ***NADPH*** ***oxidase*** ***inhibitor*** diphenyleneiodium (DPI) chloride, on the Ox- and crystal-induced expression of MCP-1 mRNA and protein. The transcription of MCP-1 mRNA in the cells was ***determined*** using real-time polymerase chain reaction. Hydrogen peroxide and 8-isoprostane were measured to investigate the involvement of ROS. Results. Exposure of NRK52E cells to Ox ions as well as the crystals resulted in increased expression of MCP-1 mRNA and production of the chemoattractant. Treatment with catalase reduced the Ox- and crystal-induced expression of both MCP-1 mRNA and protein. DPI reduced the crystal-induced ***gene*** expression and protein production but not Ox-induced ***gene*** expression and protein production. Conclusions. Exposure to Ox ions, and COM and Br crystals stimulates a ROS-mediated increase in MCP-1 mRNA expression and protein production. Reduction in ROS production, lipid peroxidation, low-density lipoprotein release, and inducible MCP-1 ***gene*** and protein in the presence of DPI indicates an involvement of ***NADPH*** ***oxidase*** in the production of ROS.

L10 ANSWER 28 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2005086320 ESBIOBASE

TITLE: Tumor promoter TPA stimulates MMP-9 secretion from human keratinocytes by activation of superoxide-producing NADPH oxidase
AUTHOR: Steinbrenner H.; Ramos M.C.; Stuhlmann D.; Mitic D.; Sies H.; Brenneisen P.
CORPORATE SOURCE: P. Brenneisen, Dept. of Biochemistry/Molec. Biol. I, Heinrich-Heine-University Dusseldorf, Universitatsstrasse 1, D-40225 Dusseldorf, Germany. E-mail: PeterBrenneisen@web.de
SOURCE: Free Radical Research, (2005), 39/3 (245-253), 54 reference(s) CODEN: FRARER ISSN: 1071-5762
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Matrix metalloproteinase-9 (MMP-9) is involved in physiological tissue remodelling ***processes*** as well as in tumor invasion and metastasis. The tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) increases MMP-9 secretion from normal human epidermal keratinocytes (NHEK) in vivo and in vitro. Here we show that the flavoprotein ***inhibitor*** diphenyleneiodinium (DPI) and the ***NADPH*** ***oxidase*** ***inhibitor*** apocynin block TPA-induced MMP-9 secretion of NHEK in vitro. Furthermore, N-acetyl-L-cysteine and L-cysteine lowered TPA-induced MMP-9 secretion, suggesting an involvement of reactive oxygen species(ROS). TPA exerts its effect on MMP-9 ***gene*** expression and secretion via the superoxide-producing enzyme ***NADPH*** ***oxidase*** : TPA rapidly stimulates generation of superoxide anion as well as ***gene*** expression of two cytosolic ***NADPH*** ***oxidase*** subunits (p47-phox and p67-phox) after 2 h, which is followed by induction of MMP-9 ***gene*** expression after 4 h. Taken together, the novel ***finding*** herein is the TPA-induced MMP-9 secretion from normal human epidermal keratinocytes through a ***NADPH*** ***oxidase*** dependent pathway.

L10 ANSWER 29 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2004290323 ESBIOBASE
TITLE: Substance P mediates AP-1 induction in A549 cells via reactive oxygen species
AUTHOR: Springer J.; Pleimes D.; Scholz F.R.; Fischer A.
CORPORATE SOURCE: E-mail: jochen.springer@charite.de
SOURCE: Regulatory Peptides, (15 JAN 2005), 124/1-3 (99-103), 20 reference(s) CODEN: REPPDY ISSN: 0167-0115
PUBLISHER ITEM IDENT.: S0167011504002368
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English
AB A common feature in asthma is the induction of reactive oxygen species (ROS) and the AP-1 transcription factor during the inflammatory ***process*** . AP-1 induction leads to an increased expression of pro-inflammatory cytokines. Also, higher levels of the pro-inflammatory neuropeptide ***substance*** P (SP) have been reported in bronchoalveolar-lavage fluid of asthmatics. Here, the role of SP on ROS induction and the downstream activation of AP-1 in A549 airway epithelial cells was investigated by dichloroflourescein-diacetate ***method*** and reporter ***gene*** assays. The SP-mediated AP-1 induction was dependent on extracellular calcium and ROS. The likely source of ROS are the mitochondria as rotenone ***inhibited*** AP-1 induction and the p47.sup.p.sup.h.sup.o.sup.x subunit of the ***NADPH*** ***oxidase*** complex, responsible for ROS generation in phagocytotic cells, was not expressed in A549 cells assayed by RT-PCR. This is consistent with results obtained from cells of murine bronchial epithelium, ***isolated*** by laser capture microdissection. In summary, this study provides evidence for an SP-mediated induction of AP-1, which may contribute to the expression of pro-inflammatory cytokines.

L10 ANSWER 30 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2004:300221 USPATFULL
TITLE: Translational profiling
INVENTOR(S): Chicz, Roman M., Belmont, MA, UNITED STATES
Tomlinson, Andrew J., Wayland, MA, UNITED STATES
Urban, Robert G., Lexington, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004236091	A1	20041125
APPLICATION INFO.:	US 2004-473127	A1	20040617 (10)
	WO 2002-US9671		20020328

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-60279495	20010328
	US 2001-60292544	20010521
	US 2001-60310801	20010808
	US 2001-60326370	20011001
	US 2001-60336780	20011204
	US 2002-60358985	20020220

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
LINE COUNT: 4964
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polypeptides representative of proteins expressed by a given cell type and isolated nucleic acids that encode the polypeptides are disclosed. The compositions and method described can be used to define a cell type at a given developmental, metabolic, or disease stage by identifying and cataloging proteins expressed in the cell. The compositions can also be used in the manufacture of therapeutics as well as in diagnostics and drug screening.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 31 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2004:232967 USPATFULL
TITLE: Effectors of innate immunity determination
INVENTOR(S): Hancock, Robert E. W., Vancouver, CANADA
Finlay, B. Brett, Richmond, CANADA
Scott, Monisha Gough, Vancouver, CANADA
Bowdish, Dawn, Vancouver, CANADA
Rosenberger, Carrie Melissa, Vancouver, CANADA
Powers, Jon-Paul Steven, Vancouver, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004180038	A1	20040916
APPLICATION INFO.:	US 2003-661471	A1	20030912 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-308905, filed on 2 Dec 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-336632P	20011203 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GRAY CARY WARE & FREIDENRICH LLP, 4365 EXECUTIVE DRIVE, SUITE 1100, SAN DIEGO, CA, 92121-2133
NUMBER OF CLAIMS: 93
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 6355
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of identifying a polynucleotide or pattern of polynucleotides

regulated by one or more sepsis or inflammatory inducing agents and inhibited by a peptide is described. A method of identifying a pattern of polynucleotide expression for inhibition of an inflammatory or septic response. The method includes contacting cells with LPS, LTA, CpG DNA and/or intact microbe or microbial components in the presence or absence of a cationic peptide; detecting a pattern of polynucleotide expression for the cells in the presence and absence of the peptide, wherein the pattern in the presence of the peptide represents inhibition of an inflammatory or septic response. Also included are compounds and agents identified by the methods of the invention. In another aspect, the invention provides methods and compounds for enhancing innate immunity in a subject.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 32 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2004:44525 USPATFULL
TITLE: Compositions and methods relating to endothelial cell signaling using the protease activated receptor (PAR1)
INVENTOR(S): Ruf, Wolfram, San Diego, CA, UNITED STATES
Riewald, Matthias, La Jolla, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004033517	A1	20040219
APPLICATION INFO.:	US 2003-418938	A1	20030418 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-374110P	20020419 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOODCOCK WASHBURN LLP, ONE LIBERTY PLACE, 46TH FLOOR, 1650 MARKET STREET, PHILADELPHIA, PA, 19103
NUMBER OF CLAIMS: 44
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 4669
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to compositions and methods based on the characterization of an endothelial cell protein C receptor (EPCR) dependent signaling by activated protein C (APC) which acts through protease activated receptor 1 (PAR1).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 33 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2004:1784 USPATFULL
TITLE: Effectors of innate immunity determination
INVENTOR(S): Hancock, Robert E.W., Vancouver, CANADA
Finlay, B. Brett, Richmond, CANADA
Gough Scott, Monisha, Vancouver, CANADA
Bowdish, Dawn, Vancouver, CANADA
Rosenberger, Carrie Melissa, Vancouver, CANADA
Steven Powers, Jon-Paul, Vancouver, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004001803	A1	20040101
APPLICATION INFO.:	US 2002-308905	A1	20021202 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-336632P	20011203 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MORGAN & FINNEGAN, L.L.P., 345 PARK AVENUE, NEW YORK, NY, 10154
NUMBER OF CLAIMS: 88
EXEMPLARY CLAIM: 1

LINE COUNT: 5838
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of identifying a polynucleotide or pattern of polynucleotides regulated by one or more sepsis or inflammatory inducing agents and inhibited by a peptide is described. A method of identifying a pattern of polynucleotide expression for inhibition of an inflammatory or septic response. The method includes contacting cells with LPS, LTA, CpG DNA and/or intact microbe or microbial components in the presence or absence of a cationic peptide; detecting a pattern of polynucleotide expression for the cells in the presence and absence of the peptide, wherein the pattern in the presence of the peptide represents inhibition of an inflammatory or septic response. Also included are compounds and agents identified by the methods of the invention. In another aspect, the invention provides methods and compounds for enhancing innate immunity in a subject.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 34 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2004:66006 USPATFULL
TITLE: DNA array sequence selection
INVENTOR(S): Lorenz, Matthias, Bethesda, MD, United States
PATENT ASSIGNEE(S): The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6706867	B1	20040316
APPLICATION INFO.:	US 2000-741238		20001219 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Horlick, Kenneth R.
ASSISTANT EXAMINER: Wilder, Cynthia
LEGAL REPRESENTATIVE: Leydig, Voit & Mayer, Ltd.
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 29 Drawing Page(s)
LINE COUNT: 23532
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods and compositions for the construction of custom cDNA microarrays. In particular, the methods involve the selection of relevant clusters based on knowledge and expression patterns using public database information and the identification of the best representative cDNA clones within the selected cluster. The methods facilitate the construction of custom microarrays suitable for use in any biotechnological art. In preferred embodiments, the present invention provides the the ImmunoChip.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 35 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2004291573 ESBIOBASE
TITLE: Paraoxonase 2 (PON2) expression is upregulated via a reduced-nicotinamide- adenine-dinucleotide-phosphate (NADPH)-oxidase-dependent mechanism during monocytes differentiation into macrophages
AUTHOR: Shiner M.; Fuhrman B.; Aviram M.
CORPORATE SOURCE: E-mail: fuhrman@tx.technion.ac.il
SOURCE: Free Radical Biology and Medicine, (15 DEC 2004), 37/12 (2052-2063), 50 reference(s)
CODEN: FRBMEH ISSN: 0891-5849
PUBLISHER ITEM IDENT.: S0891584904006926
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Paraoxonase 2 (PON2) is a member of the paraoxonases ***gene*** family. PON2 is ubiquitously present in cells, including macrophages, and

it was shown to protect against cellular oxidative stress. The aim of the present study was to analyze mechanisms involved in PON2 expression during monocyte/macrophage differentiation. PON2 expression was analyzed in vitro in THP-1 cells differentiated with 1.alpha.,25-dihydroxyvitamin D3 and in vivo in mouse peritoneal macrophages (MPM) ***isolated*** at increasing time intervals after intraperitoneal thioglycollate injection. PON2 expression (mRNA and protein) and activity gradually increased during monocyte/macrophage differentiation, up to five fold and eight fold in vitro and in vivo, respectively. This effect was associated with a gradual increase in cellular superoxide anion production. Supplementation of vitamin E to Balb/C mice ***inhibited*** the reduced nicotinamide adenine dinuleotide phosphate (***NADPH***)- ***oxidase*** -dependent increase in cellular superoxide anion production by 50% and down-regulated PON2 mRNA expression and activity by 30 and 60%, respectively. Furthermore, PON2 expression was lower by nine fold in MPM ***isolated*** from P47.sup.p.sup.h.sup.o.sup.x.sup.-.sup./.sup.- (inactive ***NADPH*** ***oxidase***) mice, in comparison to MPM from control mice. PON2 expression was found to be regulated, at least in part, by the transcription factor AP-1, as suggested by decreased JDP2 (AP-1 repressor) protein expression in the nucleus and by decreased PON2 expression in the presence of a Jun N-terminal kinase ***inhibitor*** (SP600125). The present study demonstrates, for the first time, that PON2 expression increases in monocytes during their maturation into macrophage as a result of ***NADPH*** - ***oxidase*** activation, and this ***process*** is partly regulated by the transcription factor AP-1. PON2 stimulation may represent a compensatory mechanism against the increase in cellular superoxide anion production and atherogenesis.

L10 ANSWER 36 OF 62 LIFESCI COPYRIGHT 2005 CSA on STN
ACCESSION NUMBER: 2004:79993 LIFESCI
TITLE: Cathelicidins, multifunctional peptides of the innate immunity
AUTHOR: Zanetti, M.
CORPORATE SOURCE: Dept. Biomedical Sciences and Technology University of Udine P.le Kolbe 4, I-33100 Udine, Italy; E-mail: zanetti@icgeb.trieste.it
SOURCE: Journal of Leukocyte Biology [J. Leukocyte Biol.], (20040100) vol. 75, no. 1, pp. 39-48.
ISSN: 0741-5400.
DOCUMENT TYPE: Journal
TREATMENT CODE: General Review
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Cathelicidins comprise a family of mammalian proteins containing a C-terminal cationic antimicrobial domain that becomes active after being freed from the N-terminal cathelin portion of the holoprotein. Many other members of this family have been ***identified*** since the first cathelicidin ***sequences*** were reported 10 years ago. The mature peptides generally show a wide spectrum of antimicrobial activity and, more recently, some of them have also been found to exert other biological activities. The human cathelicidin peptide LL-37 is chemotactic for neutrophils, monocytes, mast cells, and T cells; induces degranulation of mast cells; alters transcriptional responses in macrophages; stimulates wound vascularization and re-epithelialization of healing skin. The porcine PR-39 has also been involved in a variety of ***processes*** , including promotion of wound repair, induction of angiogenesis, neutrophils chemotaxis, and ***inhibition*** of the phagocyte ***NADPH*** ***oxidase*** activity, whereas the bovine BMAP-28 induces apoptosis in transformed cell lines and activated lymphocytes and may thus help with clearance of unwanted cells at inflammation sites. These multiple actions provide evidence for active participation of cathelicidin peptides in the regulation of the antimicrobial host defenses.

L10 ANSWER 37 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2004014888 ESBIOBASE

TITLE: Source of early reactive oxygen species in the apoptosis induced by transforming growth factor-.beta. in fetal rat hepatocytes
AUTHOR: Herrera B.; Murillo M.M.; Alvarez-Barrientos A.; Beltran J.; Fernandez M.; Fabregat I.
CORPORATE SOURCE: Dr. I. Fabregat, Depto. de Bioquim. y Biol. Molec., Facultad de Farmacia, Ciudad Universitaria, 28040 Madrid, Spain.
E-mail: isabelf@farm.ucm.es
SOURCE: Free Radical Biology and Medicine, (01 JAN 2004), 36/1 (16-26), 50 reference(s)
CODEN: FRBMEH ISSN: 0891-5849
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Transforming growth factor-.beta. (TGF-.beta.) induces an oxidative stress ***process*** in hepatocytes that mediates its apoptotic activity. To ***determine*** the cellular source of the early reactive oxygen species (ROS) generated by fetal rat hepatocytes in response to TGF-.beta., we used ***inhibitors*** that block different ROS-producing systems. Diphenyleneiodonium, which ***inhibits*** ***NADPH*** ***oxidase*** and other flavoproteins, completely blocked the increase in ROS induced by TGF-.beta., coincidently with an impairment of caspase-3 activation and cell death. Rotenone, an ***inhibitor*** of the NADH dehydrogenase in mitochondrial complex I, attenuated, but did not completely ***inhibit*** , ROS-production, caspase activation, and cell death mediated by TGF-.beta.. No significant protection was observed with ***inhibitors*** of other ROS-producing systems, such as cytochrome P450 (metyrapone), cyclooxygenase (indomethacin), and xanthine ***oxidase*** (allopurinol). Additional experiments have indicated that two different mechanisms could be involved in the early ROS production by TGF-.beta.. First, an inducible (cycloheximide- ***inhibited***) ***NADPH*** ***oxidase*** -like system could account for the extramitochondrial production of ROS. Second, TGF-.beta. could increase ROS by a rapid downregulation of antioxidant ***genes*** . In particular, intramitochondrial ROS would increase by depletion of MnSOD. Finally, glutathione depletion is a late event and it would be more the consequence than the cause of the increase in ROS induced by TGF-.beta..

L10 ANSWER 38 OF 62 CAPLUS
ACCESSION NUMBER: 2003:991675 CAPLUS
DOCUMENT NUMBER: 140:37984
TITLE: Alternatively spliced isoforms of NADPH oxidase NOX1-b, specifically expressed in periosteum of RA patients, cDNA cloning, and diagnostic and drug screening uses
INVENTOR(S): Kawakami, Masakatsu
PATENT ASSIGNEE(S): Yamanouchi Pharmaceutical Co., Ltd., Japan
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003104454	A1	20031218	WO 2003-JP7148	20030605

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,

BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
CA 2480619 AA 20031218 CA 2003-2480619 20030605
EP 1482034 A1 20041201 EP 2003-730849 20030605
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
US 2005124056 A1 20050609 US 2003-509622 20030605
JP 3716858 B2 20051116 JP 2004-511514 20030605
PRIORITY APPLN. INFO.: JP 2002-165612 A 20020606
JP 2003-60749 A 20030307
WO 2003-JP7148 W 20030605

AB This invention disclose an NADPH oxidase isoform NOX1-b, encoding gene and uses. Further, it disclose a ***method*** of diagnosing rheumatoid arthritis (RA) using PCR primers, and ***screening*** ***substances*** .useful for treating RA and/or arthritis deformans with the use of the novel ***NOX1*** -b oxidase ***gene*** . CDNA encoding a novel NADPH oxidase specifically expressed in periosteum of RA patients was cloned. This oxidase NOX1-b was found to be an alternatively spliced isoform of NOX1 (Mox1, GenBank AF127763). Expression of NOX1-b mRNA was found to be significantly elevated in periosteum of RA patients. NOX1-b producing cells showed a marked reactive oxygen species (ROS) prodn. activity and this was inhibited by NADPH oxidase inhibitor diphenylene iodonium chloride (DPI). Expression of COX-2 and TNF.alpha. were also elevated in NOX1-b producing cells.

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 39 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2003:265846 USPATFULL
TITLE: Nck SH3 binding peptides
INVENTOR(S): Sparks, Andrew B., Pikesville, MD, UNITED STATES
Kay, Brian K., Chapel Hill, NC, UNITED STATES
Thorn, Judith M., Carrboro, NC, UNITED STATES
Quilliam, Lawrence A., Indianapolis, IN, UNITED STATES
Der, Channing J., Chapel Hill, NC, UNITED STATES
Fowlkes, Dana M., Chapel Hill, NC, UNITED STATES
Rider, James E., Carrboro, NC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003186863	A1	20031002
APPLICATION INFO.:	US 2002-161791	A1	20020531 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-500124, filed on 8 Feb 2000, GRANTED, Pat. No. US 6432920 Division of Ser. No. US 1996-602999, filed on 16 Feb 1996, GRANTED, Pat. No. US 6184205 Continuation-in-part of Ser. No. US 1995-483555, filed on 7 Jun 1995, ABANDONED Continuation-in-part of Ser. No. US 1994-278865, filed on 22 Jul 1994, GRANTED, Pat. No. US 6303574
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MORGAN & FINNEGAN, L.L.P., 345 Park Avenue, New York, NY, 10154-0053
NUMBER OF CLAIMS: 126
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 7111
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Peptides having general and specific binding affinities for the Src homology region 3 (SH3) domains of proteins are disclosed in the present invention. In particular, SH3 binding peptides have been isolated from phage-displayed random peptide libraries which had been screened for isolates that bind to bacterial fusion proteins comprising SH3 and glutathione S-transferase (GST). Preferred peptides are disclosed which comprise a core 7-mer sequence (preferably, a consensus motif) and two or more, preferably at least six, additional amino acid residues flanking the core sequence, for a total length of 9, preferably at least 13, amino acid residues and no more than about 45 amino acid residues. Such peptides manifest preferential binding affinities for certain SH3 domains. The preferred peptides exhibit specific binding affinities for the Src-family of proteins. In vitro and in vivo results are presented

which demonstrate the biochemical activity of such peptides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 40 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2003:200808 USPATFULL
TITLE: Molecular signatures of commonly fatal carcinomas
INVENTOR(S): Su, Andrew I., La Jolla, CA, UNITED STATES
Hampton, Garret M., San Diego, CA, UNITED STATES
PATENT ASSIGNEE(S): IRM LLC, a Delaware Limited Liability Company, Hamilton HM LX, BERMUDA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003138793	A1	20030724
APPLICATION INFO.:	US 2002-167755	A1	20020610 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-297277P	20010610 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TIMOTHY L. SMITH, GENOMICS INSTITUTE OF THE, NOVARTIS RESEARCH FOUNDATION, 10675 JOHN JAY HOPKINS DRIVE, SUITE E225, SAN DIEGO, CA, 92121-1127
NUMBER OF CLAIMS: 74
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 2555
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides methods, kits, and algorithms for obtaining molecular signatures of cells based on their gene expression profiles. Devices for carrying out molecular signature analysis of unknown samples are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 41 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2003:120243 USPATFULL
TITLE: Compositions affecting programmed cell death and their use in the modification of plant development
INVENTOR(S): Flinn, Barry, Fredericton, CANADA
Lasham, Annette, Auckland, NEW ZEALAND
PATENT ASSIGNEE(S): Genesis Research and Development Corporation Limited, Auckland, NEW ZEALAND (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003082724	A1	20030501
APPLICATION INFO.:	US 2002-219220	A1	20020814 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-325932, filed on 4 Jun 1999, GRANTED, Pat. No. US 6451604
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SPECKMAN LAW GROUP, 1501 WESTERN AVE, SUITE 100, SEATTLE, WA, 98101
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 9341
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel isolated polynucleotides associated with programmed cell death and various plant developmental mechanisms are provided, together with genetic constructs comprising such sequences. Methods for the modulation of the content, structure and metabolism of plants, and particularly for the modulation of PCD and various plant developmental mechanisms in plants, are also disclosed, the methods comprising incorporating one or more of the polynucleotides or genetic constructs of the present invention into the genome of a plant.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 42 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2003:106700 USPATFULL
TITLE: Kits and methods for assessing skin health
INVENTOR(S): DePhillipo, John R., Margate, NJ, UNITED STATES
Ricciardi, Robert P., Glen Mills, PA, UNITED STATES
PATENT ASSIGNEE(S): GeneLink, Incorporated, Margate, NJ (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003073612	A1	20030417
APPLICATION INFO.:	US 2002-247935	A1	20020920 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2002-US10682, filed on 5 Apr 2002, PENDING Continuation-in-part of Ser. No. US 2001-826522, filed on 5 Apr 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-289169P	20010507 (60)
	US 2001-350517P	20011022 (60)
	US 2001-335426P	20011024 (60)
	US 2001-336815P	20011205 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AKIN GUMP STRAUSS HAUER & FELD L.L.P., ONE COMMERCE SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA, PA, 19103-7013
NUMBER OF CLAIMS: 48
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 1750
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to kits and methods for assessing skin health for a human and the human's susceptibility to skin disorders. The methods involve assessing occurrence in the human's genome of one or more polymorphisms (e.g., single nucleotide polymorphisms) that occur in one or more genes associated disclosed herein and that are associated with a disorder in humans. Preferred assessment and scoring methods are disclosed, as are kits for performing the methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 43 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2003251919 ESBIOBASE
TITLE: Withdrawal of cerivastatin induces monocyte chemoattractant protein 1 and tissue factor expression in cultured vascular smooth muscle cells
AUTHOR: Brandes R.P.; Beer S.; Ha T.; Busse R.
CORPORATE SOURCE: Dr. R.P. Brandes, Inst. fur Kardiovaskulare Physiol., Klinikum der J.W. Goethe-Universitat, Theodor-Stern-Kai 7, D-60596 Frankfurt am Main, Germany.
E-mail: r.brandes@em.uni-frankfurt.de
SOURCE: Arteriosclerosis, Thrombosis, and Vascular Biology, (2003), 23/10 (1794-1800), 26 reference(s)
CODEN: ATVBFA ISSN: 1079-5642
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Objective - The withdrawal of 3-hydroxy-3-methylglutaryl-coenzyme A-reductase ***inhibitors*** (statins) deteriorates endothelial function. We ***determined*** in vascular smooth muscle cells whether statin withdrawal leads to the expression of proinflammatory ***genes*** involved in the development and progression of arteriosclerosis. ***Methods*** and Results - The withdrawal of cerivastatin from pretreated vascular smooth muscle cells induced an increase in monocyte chemoattractant protein 1 (MCP-1) and tissue factor

(TF) mRNA expression and enhanced MCP-1 secretion as well as cell surface TF activity. In the presence of cerivastatin, this effect was mimicked by geranylgeranyl pyrophosphate or mevalonate. Withdrawal-induced MCP-1 expression was sensitive to PD98059, SB203580, and diphenylene iodonium, suggesting an involvement of extracellular signal-regulated kinase 1/2, p38 mitogen-activated protein kinase, and the ***NADPH*** ***oxidase*** . Withdrawal increased the activity of extracellular signal-regulated kinase 1/2 and p38 mitogen-activated protein kinase and enhanced radical generation. Because the latter effect may result from an Rac-mediated activation of the ***NADPH*** ***oxidase*** , the effect of withdrawal on Rac translocation was studied. Statin treatment induced an increase in Rac-1 content in the cytoplasm. On withdrawal, however, an "overshoot" translocation of Rac to the plasma membrane occurred. Conclusions - These observations suggest that statin withdrawal results in the activation of Rac and enhanced oxidative stress. The subsequent activation of redox-activated signal-transduction cascades results in the expression of MCP-1 and TF.

L10 ANSWER 44 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V.
on STN DUPLICATE
ACCESSION NUMBER: 2003058555 ESBIOBASE
TITLE: Endothelin-1 increases vascular superoxide via endothelin.sub.A-NADPH oxidase pathway in low-renin hypertension
AUTHOR: Li L.; Fink G.D.; Watts S.W.; Northcott C.A.; Galligan J.J.; Pagano P.J.; Chen A.F.
CORPORATE SOURCE: Dr. A.F. Chen, Department of Pharmacology, B403 Life Sciences Building, Michigan State University, East Lansing, MI 48824-1317, United States.
E-mail: chenal@msu.edu
SOURCE: Circulation, (25 FEB 2003), 107/7 (1053-1058), 40 reference(s)
CODEN: CIRCAZ ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background - Angiotensin II-induced hypertension is associated with NAD(P)H ***oxidase*** -dependent superoxide production in the vessel wall. Vascular superoxide level is also increased in deoxycorticosterone acetate (DOCA)-salt hypertension, which is associated with a markedly depressed plasma renin activity because of sodium retention. However, the mechanisms underlying superoxide production in low-renin hypertension are undefined. ***Methods*** and Results - This study investigated (1) whether and how endothelin-1 (ET-1), which is increased in DOCA-salt hypertensive rats, contributes to arterial superoxide generation and (2) the effect of ***gene*** transfer of manganese superoxide dismutase and endothelial nitric oxide synthase. Both superoxide and ET-1 levels were significantly elevated in carotid arteries of DOCA-salt rats compared with that of the sham-operated controls. ET-1 concentration-dependently stimulated superoxide production in vitro in carotid arteries of normotensive rats. The increase in arterial superoxide in both ET-1-treated normotensive and DOCA-salt rats was reversed by a selective ET.sub.A receptor ***antagonist*** , ABT-627, the flavoprotein ***inhibitor*** diphenyleneiodonium, and the ***NADPH*** ***oxidase*** ***inhibitor*** apocynin but not by the nitric oxide synthase ***inhibitor*** N.sup.&.sup.o.sup.m.sup.e.sup.g.sup.a.sup.;-L-arginine methyl ester or the xanthine ***oxidase*** ***inhibitor*** allopurinol. Furthermore, in vivo blockade of ET.sub.A receptors significantly reduced arterial superoxide levels, with a concomitant decrease of systolic blood pressure in DOCA-salt rats. Ex vivo ***gene*** transfer of manganese superoxide dismutase or endothelial nitric oxide synthase also suppressed superoxide levels in carotid arteries of DOCA-salt rats. Conclusions - These ***findings*** suggest that ET-1 augments vascular superoxide production at least in part via an ET.sub.A/ ***NADPH*** ***oxidase*** pathway in low-renin mineralocorticoid hypertension.

L10 ANSWER 45 OF 62 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.
on STN DUPLICATE 9

ACCESSION NUMBER: 2003-0172836 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2003 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Potential thrombophilic mutations/polymorphisms in patients with no flow-limiting stenosis after myocardial infarction
AUTHOR: FRENCH John K.; VAN DE WATER Neil S.; SUTTON Timothy M.; LUND Mayanna; WANZHEN GAO; MCDOWELL Joanne; LIU-STRATTON Yiwen; POHORENCE Jeanette; SZYMANSKI Diane; GOLDSCHMIDT-CLERMONT Pascal; WHITE Harvey D.; BROWETT Peter J.; COOKE Glen
CORPORATE SOURCE: Department of Molecular Medicine, University of Auckland, Auckland, New Zealand; Haematology Department, Auckland Hospital, Auckland, New Zealand; Cardiology Department, Green Lane Hospital, Auckland, New Zealand; Heart and Lung Institute, Ohio State University, Columbus, Ohio, United States; Division of Cardiology, Department of Medicine Duke University Medical Center, Durham, NC, United States
SOURCE: The American heart journal, (2003), 145(1), 118-124, 42 refs.
ISSN: 0002-8703 CODEN: AHJOA2
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-2057, 354000104161970180
AN 2003-0172836 PASCAL
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AB Background Although inherited thrombophilias are more common in patients with venous thromboembolism, their influence on the development of myocardial infarction (MI) requires clarification. ***Methods*** and Results To ***determine*** whether there are increased frequencies of mutations/polymorphisms in 14 ***genes*** potentially causing thrombophilia in patients with no flow-limiting stenoses after MI compared with patients with >= 1 flow-limiting stenosis of >50%, we studied 395 patients (60 with no flow-limiting stenosis) who underwent angiography at approximately 1 month. The mutations/polymorphisms studied included Factor V Leiden, prothrombin variant G20210A, .beta.-fibrinogen 448 (G/A), endothelial protein C receptor (23-base pair insertion), methyl tetrahydrofolate reductase 677 (C/T), platelet glycoprotein IIIa PIA1/A2, plasminogen activator ***inhibitor*** -1 4G/5G, angiotensin II type 1 receptor (A/C), hemochromatosis ***gene*** 282 (G/A), nitric oxide synthase (NOS) (3 forms: eNOS, eNOS3, eNOS4), p22 phox of ***NADPH*** ***oxidase*** C242T, and angiotensin-converting enzyme insertion/deletion polymorphism. The frequencies of Factor V Leiden and the .beta.-fibrinogen 448 A allele were higher in patients with no flow-limiting stenosis than in patients with >=1 stenosis (11.7% vs 3.6%, odds ratio [OR] 3.6, 95% CI 1.3-9.4, P =.015; and 42% vs 27%, OR 2.0, 95% CI 1.1-3.5, P =.018, respectively), and there was a trend toward an increased frequency of prothrombin variant G20210A (6.7% vs 2.1%, OR 3.4, 95% CI 0.95-11.8, P =.069). However, in patients with no flow-limiting stenosis after MI the frequencies of the other ***gene*** mutations/polymorphisms were not increased. Also, there were no significant interactions between any of these 14 mutation/polymorphisms, major cardiovascular risk factors, and the absence of any flow-limiting stenosis, except for Factor V Leiden and hypertension (OR 6.34, 95% CI 2.67-100, P =.004). Conclusions Patients with no flow-limiting stenosis after MI had increased frequencies of 2 inherited thrombophilias (Factor V Leiden and .beta.-fibrinogen 448 A allele), and there was a trend toward an increased frequency of prothrombin variant G20210A compared with patients with >=1 stenosis. These data suggest that polymorphisms/mutations in some ***gene*** products influencing coagulation may influence the pathogenesis of MI.

L10 ANSWER 46 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:51656 CAPLUS
DOCUMENT NUMBER: 136:97270
TITLE: Screening method for identifying genes in human neutrophil cells involved in colony-stimulating factor

mediated inhibition of cell death
INVENTOR(S): Cotter, Tom; Hayes, Ian; Murphy, Finbarr; Seery, Liam
PATENT ASSIGNEE(S): Eirx Therapeutics Limited, Ire.
SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002004657	A2	20020117	WO 2001-GB3101	20010709
WO 2002004657	A3	20030116		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1299558	A2	20030409	EP 2001-947681	20010709
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003190650	A1	20031009	US 2003-332130	20030519

PRIORITY APPLN. INFO.: GB 2000-16692 A 20000707
US 2000-254459P P 20001208
WO 2001-GB3101 W 20010709

AB The present invention uses human neutrophil as a model system for the study of granulocyte macrophage colony-stimulating factor (GM-CSF) mediated inhibition of apoptosis and discloses a method for identifying genes involved in modulating the transition of a cell between a non-apoptotic state and an apoptotic state in this system. In particular, the method comprises the steps of: (a) exposing the cell to an inhibitor of GM-CSF mediated inhibition of apoptosis; and (b) exposing the cell to one or more agents which increase tyrosine phosphorylation; and (c) placing the cell in conditions which permit it to undergo spontaneous apoptosis; and (d) monitoring the levels of expression of the gene products in the cell; and (e) identifying genes whose expression has been increased, decreased or modified by using microassay. The invention further discloses that the system provides the means to characterize the mol. mechanisms of apoptosis in neutrophils, in particular, the GM-CSF mediated inhibition of apoptosis.

L10 ANSWER 47 OF 62 USPATFULL on STN
ACCESSION NUMBER: 2002:202061 USPATFULL
TITLE: Nck SH3 binding peptides
INVENTOR(S): Sparks, Andrew B., Baltimore, MD, United States
Kay, Brian K., Madison, WI, United States
Thorn, Judith M., Galesburg, IL, United States
Quilliam, Lawrence A., Indianapolis, IN, United States
Der, Channing J., Chapel Hill, NC, United States
Fowlkes, Dana M, Chapel Hill, NC, United States
Rider, James E, Eagan, MN, United States
PATENT ASSIGNEE(S): Cytogen Corporation, Princeton, NJ, United States (U.S. corporation)
University of North Carolina at Chapel Hill, Chapel Hill, NC, United States (U.S. corporation)

NUMBER	KIND	DATE
PATENT INFORMATION: US 6432920	B1	20020813
APPLICATION INFO.: US 2000-500124		20000208 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1996-602999, filed on 16 Feb 1996, now patented, Pat. No. US 6184205
Continuation-in-part of Ser. No. US 1995-483555, filed on 7 Jun 1995, now abandoned Continuation-in-part of Ser. No. US 1994-278865, filed on 22 Jul 1994, now

patented, Pat. No. US 6303574
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Ponnaluri, Padmashri
LEGAL REPRESENTATIVE: Morgan & Finnegan, LLP
NUMBER OF CLAIMS: 2
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Figure(s); 16 Drawing Page(s)
LINE COUNT: 6366
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Peptides having general and specific binding affinities for the Src homology region 3 (SH3) domains of proteins are disclosed in the present invention. In particular, SH3 binding peptides have been isolated from phage-displayed random peptide libraries which had been screened for isolates that bind to bacterial fusion proteins having an SH3 domain and glutathione S-transferase (GST). Preferred peptides are disclosed which comprise a core 7-mer sequence (preferably, a consensus motif) and two or more, preferably at least six, additional amino acid residues flanking the core sequence, for a total length of 9, preferably at least 13, amino acid residues and no more than about 45 amino acid residues. Such peptides manifest preferential binding affinities for certain SH3 domains. The preferred peptides exhibit specific binding affinities for the Src-family of proteins. In vitro and in vivo results are presented which demonstrate the biochemical activity of such peptides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 48 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2002135702 ESBIOBASE
TITLE: Phosphorylation of p47.sup.p.sup.h.sup.o.sup.x sites by PKC .alpha., .beta.II, .delta., and .zeta.: Effect on binding to p22.sup.p.sup.h.sup.o.sup.x and on NADPH oxidase activation
AUTHOR: Fontayne A.; Dang P.M.-C.; Gougerot-Pocidalo M.-A.; El Benna J.
CORPORATE SOURCE: J. El Benna, INSERM U-479, CHU X. Bichat, 16 rue Henri Huchard, 75018 Paris, France.
E-mail: benna@bichat.inserm.fr
SOURCE: Biochemistry, (18 JUN 2002), 41/24 (7743-7750), 35 reference(s)
CODEN: BICHAW ISSN: 0006-2960
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Production of superoxide anions by the multicomponent enzyme of human neutrophil ***NADPH*** ***oxidase*** is accompanied by extensive phosphorylation of p47.sup.p.sup.h.sup.o.sup.x, one of its cytosolic components. p47.sup.p.sup.h.sup.o.sup.x is an excellent substrate for protein kinase C (PKC), but the respective contribution of each PKC isoform to this ***process*** is not clearly defined. In this study, we found that PKC isoforms known to be present in human neutrophils (PKC .alpha., .beta., .delta., and .zeta.) phosphorylate p47.sup.p.sup.h.sup.o.sup.x in a time- and concentration-dependent manner, with apparent K.sub.m values of 10.33, 3.37, 2.37, and 2.13 .mu.M for PKC .alpha., .beta.II, .delta., and .zeta., respectively. Phosphopeptide mapping of p47.sup.p.sup.h.sup.o.sup.x showed that, as opposed to PKC .zeta., PKC .alpha., .beta.II, and .delta. are able to phosphorylate all the major PKC sites. The use of p47.sup.p.sup.h.sup.o.sup.x mutants ***identified*** serines 303, 304, 315, 320, 328, 359, 370, and 379 as targets of PKC .alpha., .beta.II, and .delta.. Comparison of the intensity of phosphopeptides suggests that Ser 328 is the most phosphorylated serine. The ability of each PKC isoform to induce p47.sup.p.sup.h.sup.o.sup.x to associate with p22.sup.p.sup.h.sup.o.sup.x was tested by using an overlay technique; the results showed that all the PKC isoforms that were studied induce p47.sup.p.sup.h.sup.o.sup.x binding to the cytosolic fragment of p22.sup.p.sup.h.sup.o.sup.x. In addition, PKC .alpha., .beta.II, .delta., and .zeta. were able to induce production of superoxide anions in a

cell-free system using ***recombinant*** cytosolic proteins. Surprisingly, PKC .zeta., which phosphorylates a subset of selective p47.sup.p.sup.h.sup.o.sup.x sites, induced stronger activation of the ***NADPH*** ***oxidase*** . Taken together, these results suggest that PKC .alpha., .beta.II, .delta., and .zeta. expressed in human neutrophils can individually phosphorylate p47.sup.p.sup.h.sup.o.sup.x and induce both its translocation and ***NADPH*** ***oxidase*** activation. In addition, phosphorylation of some serines could have an ***inhibitory*** effect on ***oxidase*** activation.

L10 ANSWER 49 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:472975 CAPLUS
DOCUMENT NUMBER: 135:72117
TITLE: Analysis of early processes in reactive oxygen species induced apoptosis and identification of target genes for therapeutic use
INVENTOR(S): Cotter, Tom; Hayes, Ian
PATENT ASSIGNEE(S): Eirx Therapeutics Ltd., Ire.
SOURCE: PCT Int. Appl., 120 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001046468	A2	20010628	WO 2000-IB2054	20001221
WO 2001046468	A3	20020613		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2003180735	A1	20030925	US 2003-168448	20030227

PRIORITY APPLN. INFO.: GB 1999-30255 A 19991221
US 1999-173199P P 19991227
WO 2000-IB2054 W 20001221

AB A method of analyzing early events in apoptosis induced by reactive oxygen species and the genes induced early in the process is described. The genes that are induced early in the process or their products may be useful as targets for therapeutic regulation of apoptosis. Apoptosis was studied in human neutrophils. The process began approx. 6 h after sample collection and was accompanied by the generation of the peroxide anion and hydrogen peroxide. Inhibition of NADPH oxidase blocked apoptosis. Anal. of gene expression in the period before apoptosis became detectable found that a no. of genes known to be involved in apoptosis were induced very early in the process.

L10 ANSWER 50 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2001256742 ESBIOBASE
TITLE: Oxidative stress in scleroderma: Maintenance of scleroderma fibroblast phenotype by the constitutive up-regulation of reactive oxygen species generation through the NADPH oxidase complex pathway
AUTHOR: Sambo P.; Baroni S.S.; Luchetti M.; Paroncini P.; Dusi S.; Orlandini G.; Gabrielli A.
CORPORATE SOURCE: Dr. A. Gabrielli, Istituto di Clinica Medica Generale, Ematologia ed Immunologia Clinica, Via Tronto, 10, 60020 Ancona, Italy.
E-mail: a.gabrielli@popcsi.unian.it
SOURCE: Arthritis and Rheumatism, (2001), 44/11 (2653-2664), 42 reference(s)
CODEN: ARHEAW ISSN: 0004-3591
DOCUMENT TYPE: Journal; Article

COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Objective. To explore the role of reactive oxygen species (ROS) in the in vitro activation of skin fibroblasts from patients with systemic sclerosis (SSc). ***Methods*** . Fibroblasts were obtained from involved skin of patients with limited or diffuse SSc. Oxidative activity imaging in living cells was carried out using confocal microscopy. Levels of O.sub.2.sup.- and H.sub.2O.sub.2 released from fibroblasts were estimated by the superoxide dismutase (SOD)- ***inhibitable*** cytochrome c reduction and homovanilic acid assays, respectively. To verify ***NADPH*** ***oxidase*** activation, the light membrane of fibroblasts was immunoblotted with an anti-p47.sup.p.sup.h.sup.o.sup.x-specific antibody. Fibroblasts were stimulated with various cytokines and growth factors to ***determine*** whether any of these factors modulate ROS generation. Cell proliferation was estimated by .sup.3H-thymidine incorporation. Northern blot analysis was used to study .alpha.1 and .alpha.2 type I collagen ***gene*** expression. Results. Unstimulated skin fibroblasts from SSc patients released more O.sub.2.sup.- and H.sub.2O.sub.2 in vitro through the ***NADPH*** ***oxidase*** complex pathway than did normal fibroblasts, since incubation of SSc fibroblasts with diphenylene iodonium, a flavoprotein ***inhibitor*** , suppressed the generation of ROS. This suppression was not seen with rotenone, a mitochondrial ***oxidase*** ***inhibitor*** , or allopurinol, a xanthine ***oxidase*** ***inhibitor*** . Furthermore, the cytosolic component of ***NADPH*** ***oxidase*** , p47.sup.p.sup.h.sup.o.sup.x, was translocated to the plasma membrane of resting SSc fibroblasts. A transient increase in ROS production was induced in normal but not in SSc fibroblasts by interleukin-1.beta. (IL-1.beta.), platelet-derived growth factor type BB (PDGF-BB), transforming growth factor .beta.1 (TGF.beta.1), and H.sub.2O.sub.2. Treatment of normal and SSc fibroblasts with tumor necrosis factor .alpha. (TNF.alpha.), IL-2, IL-4, IL-6, IL-10, interferon-.alpha. (IFN.alpha.), IFN.gamma., granulocyte-macrophage colony-stimulating factor (GM-CSP), G-CSF, or connective tissue growth factor (CTGF) had no effect on ROS generation. Constitutive ROS production by SSc fibroblasts was not ***inhibited*** when these cells were treated with catalase, SOD, IL-1 receptor ***antagonist*** , or antibodies blocking the effect of TGF.beta.1, PDGF-BB, and other agonists (IL-4, IL-6, TNF.alpha., CTGF). In contrast, treatment of SSc fibroblasts with the membrane-permeant antioxidant N-acetyl-L-Cysteine ***inhibited*** ROS production, and this was accompanied by decreased proliferation of these cells and down-regulation of .alpha.1(I) and .alpha.2(I) collagen messenger RNA. Conclusion. The constitutive intracellular production of ROS by SSc fibroblasts derives from the activation of an ***NADPH*** ***oxidase*** -like system and is essential to fibroblast proliferation and expression of type I collagen ***genes*** in SSc cells. Our results also exclude O.sub.2.sup.-, H.sub.2O.sub.2, IL-1.beta., TGF.beta.1, PDGF-BB, IL-4, IL-6, TNF.alpha., or CTGF as mediators of a positive, autocrine feedback mechanism of ROS generation.

L10 ANSWER 51 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2001119464 ESBIOBASE
TITLE: Induction of plant gp91 phox homolog by fungal cell wall, arachidonic acid, and salicylic acid in potato
AUTHOR: Yoshioka H.; Sugie K.; Park H.-J.; Maeda H.; Tsuda N.; Kawakita K.; Doke N.
CORPORATE SOURCE: H. Yoshioka, Plant Pathology Laboratory, Grad. Sch. of Bioagricultural Sci., Nagoya University, Chikusa, Nagoya 464-8601, Japan.
E-mail: hyoshiok@agr.nagoya-u.ac.jp
SOURCE: Molecular Plant-Microbe Interactions, (2001), 14/6 (725-736), 87 reference(s)
CODEN: MPMIEL ISSN: 0894-0282
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The oxidative burst has been suggested to be a primary event responsible for triggering the cascade of defense responses in various plant species against infection with avirulent pathogens or pathogen-derived elicitors. The molecular mechanisms of rapid production of active oxygen species (AOS), however, are not well known. We ***isolated*** homologs of gp91 phox, a plasma membrane protein of the neutrophil ***NADPH*** ***oxidase*** , from a potato cDNA library. Molecular cloning of the cDNA showed that there are two isogenes, designated StrbohA and StrbohB, respectively. The RNA gel blot analyses showed that StrbohA was constitutively expressed at a low level, whereas StrbohB was induced by hyphal wall components (HWC elicitor) from Phytophthora infestans in potato tubers. Treatment of potato tubers with HWC elicitor caused a rapid but weak transient accumulation of H.sub.2O.sub.2 (phase I), followed by a massive oxidative burst 6 to 9 h after treatment (phase II). Diphenylene iodonium (DPI), an ***inhibitor*** of the neutrophil ***NADPH*** ***oxidase*** , blocked both bursts, whereas pretreatment of the protein synthesis ***inhibitor*** cycloheximide with the tuber abolished only the second burst. These results suggest that the expression of StrbohA and StrbohB contributes to phase I and II bursts, respectively. The same is true for arachidonic acid, a lipid component of P. infestans-stimulated biphasic oxidative burst, whereas an endogenous signaling molecule, salicylic acid, only induced a weak phase II burst. Both molecules induced the StrbohB expression, which is in agreement with the second burst. To characterize the signal transduction pathway leading to the oxidative burst, we examined the role of protein phosphorylation in HWC-stimulated StrbohB ***gene*** expression. K252a and staurosporine, two protein kinase ***inhibitors*** , blocked the transcript accumulation. Two ***inhibitors*** of extracellular Ca.sup.2.sup.+ movement, however, did not abolish the transcript accumulation of StrbohB, suggesting that certain calcium-independent protein kinases are involved in the ***process*** of StrbohB ***gene*** expression. Additionally, we examined a causal relationship between the oxidative burst and expression of defense ***genes*** induced by the HWC elicitor. The transcript accumulation of ***genes*** related to sesquiterpenoid phytoalexin synthesis (lubimin and rishitin) and phenylpropanoid pathway was ***inhibited*** slightly by the DPI treatment, suggesting that the oxidative burst is not essential to activate these ***genes*** . Interestingly, the concomitant presence of DPI with the elicitor resulted in an increase in lubimin accumulation and a decrease in rishitin accumulation. Because it is known that lubimin is metabolized into rishitin via oxylubimin, we propose that AOS mediates the synthesis of rishitin from lubimin.

L10 ANSWER 52 OF 62 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2000-0478038 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2000 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Cytochrome P-450 enzymes and FM03 contribute to the disposition of the antipsychotic drug perazine in vitro
AUTHOR: STOERMER E.; BROCKMOELLER J.; ROOTS I.; SCHMIDER J.
CORPORATE SOURCE: Humboldt-University Berlin, Institute of Clinical Pharmacology, Schumannstr. 20/21, 10098 Berlin, Germany, Federal Republic of; Pfizer Inc., P.O. Box 8030, Groton, CT 06340-8030, United States
SOURCE: Psychopharmacologia, (2000), 151(4), 312-320, refs. 1 p.1/4
ISSN: 0033-3158
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
AVAILABILITY: INIST-1761, 354000092134560030
AN 2000-0478038 PASCAL
CP Copyright .COPYRGT. 2000 INIST-CNRS. All rights reserved.
AB Rationale: Perazine (PER) is a phenothiazine antipsychotic drug frequently used in Germany that undergoes extensive metabolism. Objectives and ***methods*** : To anticipate metabolic drug interactions and to explore the relevance of polymorphisms of metabolic

enzymes, perazine-N-demethylation and perazine-N-oxidation were investigated in vitro using human liver microsomes and cDNA expressed enzymes. Results: CYP3A4 and CYP2C9 were ***identified*** as the major enzymes mediating PER-N-demethylation. At 10 .mu.M PER, a concentration consistent with anticipated in vivo liver concentrations, CYP3A4 and CYP2C9 contributed 50% and 35%, respectively, to PER-N-demethylation. With increasing PER concentrations, contribution of CYP2C9 decreased and CYP3A4 became more important. In human liver microsomes, PER-N-demethylation was ***inhibited*** by ketoconazole (>40%) and sulfaphenazole (16%). Allelic variants of ***recombinant*** CYP2C9 showed differences in PER-N-demethylase activity. The wild type allele CYP2C9.sup.* 1 was the most active variant. Maximal activities of CYP2C9.sup.*2 and CYP2C9.sup.*3 were 88% and 18%, respectively, compared to the wild type activity. Perazine-N-oxidation was mainly mediated by FMO3. In the absence of ***NADPH*** , heat treatment of microsomes abolished PER-N- ***oxidase*** activity. Methimazole ***inhibited*** PER-N-oxidation, while CYP specific ***inhibitors*** had no ***inhibitory*** effect. Perazine is a potent ***inhibitor*** of dextromethorphan-O-demethylase, S-mephenytoin-hydroxylase, alprazolam-4-hydroxylase, phenacetin-O-deethylase and tolbutamide-hydroxylase activity in human liver microsomes. Conclusions: Alterations in the activity of CYP3A4, CYP2C9 and FMO3 through genetic polymorphisms, enzyme induction or ***inhibition*** bear the potential to cause clinically significant changes in perazine clearance. PER may alter the clearance of coadministered ***compounds*** metabolized by CYP2D6, CYP2C19, CYP2C9, CYP3A4 and CYP1A2.

L10 ANSWER 53 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V.
on STN DUPLICATE
ACCESSION NUMBER: 1999210732 ESBIOBASE
TITLE: Angiotensin II-induced superoxide anion generation in human vascular endothelial cells. Role of membrane-bound NADH-/NADPH-oxidases
AUTHOR: Zhang H.; Schmeisser A.; Garlichs C.D.; Plotze K.; Damme U.; Mugge A.; Daniel W.G.
CORPORATE SOURCE: H. Zhang, Department of Cardiology, Medical Clinic II, Friedrich-Alexander-University, Schwabachanlage 10, D-91054 Bochum, Germany.
E-mail: zhh86@hotmail.com
SOURCE: Cardiovascular Research, (1999), 44/1 (215-222), 36 reference(s)
CODEN: CVREAU ISSN: 0008-6363
PUBLISHER ITEM IDENT.: S0008636399001832
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background: Angiotensin II (ANG II) mediated hypertension accelerates atherosclerosis (AS) and thereby increases the incidence of myocardial infarction (MI). On the other hand, superoxide anion (O.sub.2.sup.-) is involved in the modification of low density lipoproteins, ***inhibition*** of prostacyclin (PGI.sub.2) formation and breakdown of nitric oxide. These events finally lead to rapid progression of AS and MI. In the present study, we investigate whether ANG II can induce O.sub.2.sup.- release from human vascular endothelial cells (HVECs) and the possible mechanisms involved. ***Methods*** and Results: The expression of ANG receptors subtype-1 (AT-1) and subtype-2 (AT-2) were ***identified*** by using reverse transcription polymerase chain reaction and ***sequence*** analysis. The O.sub.2.sup.- production was dose-dependently increased in HVECs treated with ANG II (10.sup.-.sup.7-10.sup.-.sup.9 M) and with a maximum rate after 1 h of incubation. This event was significantly ***inhibited*** by pretreatment of cells with the specific AT-1 blocker losartan (10.sup.-.sup.7 M) and to a lesser extent by the specific AT-2 receptor blocker PD123319 (10.sup.-.sup.7 M). The combined incubation of both receptor blockers was even more effective. In addition, our lucigenin-enhanced chemiluminescence assay showed that the activity of plasma membrane-bound NADH-/ ***NADPH*** - ***oxidases*** derived from ANG II-treated cells was also significantly increased, this effect was reduced in cells pretreated with losartan or to lesser extent by

PD123319. However, the activity of xanthine ***oxidase*** remained unchanged in response to ANG II. Furthermore, the basal O.sub.2.sup.- release from HVECs was ***inhibited*** in cells treated with angiotensin-converting enzyme (ACE) ***inhibitor*** , Lisinopril (10.sup.-.sup.6 M), and this event could be reversed by ANG II. Conclusion: ANG II induces O.sub.2.sup.- release in HVECs via activation of membrane-bound NADH-/ ***NADPH*** - ***oxidases*** , an effect, that is mediated by both AT-1 and AT-2 receptors. This suggests that acceleration of AS and MI in ANG II-mediated hypertension may at least be due to ANG II-induced O.sub.2.sup.- generation from vascular endothelial cells. In this case, the ACE ***inhibitors*** and the ANG receptor ***antagonists*** may act as causative 'antioxidants'. Copyright (C) 1999 Elsevier Science B.V.

L10 ANSWER 54 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 1998102154 ESBIOBASE
TITLE: Mammalian thioredoxin reductase is irreversibly inhibited by dinitrohalobenzenes by alkylation of both the redox active selenocysteine and its neighboring cysteine residue
AUTHOR: Nordberg J.; Zhong L.; Holmgren A.; Arner E.S.J.
CORPORATE SOURCE: E.S.J. Arner, Med. Nobel Inst. for Biochemistry I, Med. Biochemistry/Biophysics Dept., Karolinska Institutet, S-171 77 Stockholm, Sweden. E-mail: elias.arner@mbb.ki.se
SOURCE: Journal of Biological Chemistry, (01 MAY 1998), 273/18 (10835-10842), 45 reference(s) CODEN: JBCHA3 ISSN: 0021-9258
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The immunostimulatory dinitrohalobenzene ***compound*** 1-chloro-2,4-dinitrobenzene (DNCB) irreversibly ***inhibits*** mammalian thioredoxin reductase (TrxR) in the presence of ***NADPH*** , inducing an ***NADPH*** ***oxidase*** activity in the modified enzyme (Arner, E. S. J., Bjornstedt, M., and Holmgren, A. (1995) J. Biol. Chem. 270, 3479-3482). Here we have further analyzed the reactivity with the enzyme of DNCB and analogues with varying immunomodulatory properties. We have also ***identified*** the reactive residues in bovine thioredoxin reductase, recently discovered to be a selenoprotein. We found that 4-vinylpyridine competed with DNCB for inactivation of TrxR, with DNCB being about 10 times more efficient, and only alkylation with DNCB but not with 4-vinylpyridine induced an ***NADPH*** ***oxidase*** activity. A number of nonsensitizing DNCB analogues neither inactivated the enzyme nor induced any ***NADPH*** ***oxidase*** activity. The ***NADPH*** ***oxidase*** activity of TrxR induced by dinitrohalobenzenes generated superoxide, as detected by reaction with epinephrine (the adrenochrome ***method***). Addition of superoxide dismutase quenched this reaction and also stimulated the ***NADPH*** ***oxidase*** activity. By peptide analysis using mass spectrometry and Edman degradation, both the cysteine and the selenocysteine in the conserved carboxyl-terminal ***sequence*** Gly-Cys-Sec-Gly (where Sec indicates selenocysteine) were ***determined*** to be dinitrophenyl-alkylated upon incubation of native TrxR with ***NADPH*** and DNCB. A model for the interaction between TrxR and dinitrohalobenzenes is proposed, involving a functional FAD in the alkylated TrxR generating an anion nitroradical in a dinitrophenyl group, which in turn reacts with oxygen to generate superoxide. Production of reactive oxygen species and ***inhibited*** reduction of thioredoxin by the modified thioredoxin reductase after reaction with dinitrohalobenzenes may play a major role in the inflammatory reactions provoked by these ***compounds*** .

L10 ANSWER 55 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 1998118308 ESBIOBASE
TITLE: Mutational analysis of novel effector domains in Rac1 involved in the activation of nicotinamide adenine

dinucleotide phosphate (reduced) oxidase
AUTHOR: Toporik A.; Gorzalczany Y.; Hirshberg M.; Pick E.; Lotan O.
CORPORATE SOURCE: O. Lotan, Department of Human Microbiology, Sackler School of Medicine, Tel Aviv University, Tel Aviv 69978, Israel.
SOURCE: Biochemistry, (19 MAY 1998), 37/20 (7147-7156), 74 reference(s)
CODEN: BICHAW ISSN: 0006-2960
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The small molecular weight GTP-binding protein Rac (1 or 2) is an obligatory participant in the activation of the superoxide-generating ***NADPH*** ***oxidase*** . Active ***NADPH*** ***oxidase*** can be reconstituted in a cell-free system, consisting of phagocyte-derived membranes, containing cytochrome b559, and the ***recombinant*** cytosolic proteins p47-phox, p67-phox, and Rac, supplemented with an anionic amphiphile as an activator. The cell-free system was used before for the analysis of structural requirements of individual components participating in the assembly of ***NADPH*** ***oxidase*** . In earlier work, we mapped four previously unidentified domains in Rac1, encompassing residues 73-81 (a), 103-107 (b), 123-133 (c), and 163169 (d), as important for cell-free ***NADPH*** ***oxidase*** activation. The domains were defined by assessing the activation ***inhibitory*** effect of a series of overlapping peptides, spanning the entire length of Rac1 Joseph, G., and Pick, E. (1995) J. Biol. Chem. 270, 29079-29082 . We now used the construction of Rac1/H-Ras chimeras, domain deletion, and point mutations, to ascertain the functional relevance of three domains (b, c, and d) predicted by 'peptide walking' and to ***determine*** the importance of specific residues within these domains. This ***methodology*** firmly establishes the involvement of domains b and d in the activation of ***NADPH*** ***oxidase*** by Rac1 and ***identifies*** H103 and K166, respectively, as residues critical for the effector function of these two domains. The functional significance of domain c (insert region) could not be confirmed, as shown by the minor effect of deleting this domain on ***NADPH*** ***oxidase*** activation. Analysis of the three-dimensional structure of Rac1 reveals that residues H103 and K166 are exposed on the surface of the molecule. Modeling of the activity-impairing point mutations suggests that the effect on the ability to activate ***NADPH*** ***oxidase*** depends on the side chains of the mutated amino acids and not on changes in the global structure of the protein. In conclusion, we demonstrate the existence of two novel effector sites in Rac1, necessary for supporting ***NADPH*** ***oxidase*** activation, supplementing the canonical N-terminal effector region.

L10 ANSWER 56 OF 62 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 15
ACCESSION NUMBER: 1996:532834 SCISEARCH
THE GENUINE ARTICLE: UX943
TITLE: Assembly of the human neutrophil NADPH oxidase involves binding of p67(phox) and flavocytochrome b to a common functional domain in p47(phox)
AUTHOR: DeLeo F R (Reprint); Ulman K V; Davis A R; Jutila K L; Quinn M T
CORPORATE SOURCE: MONTANA STATE UNIV, DEPT VET MOL BIOL, BOZEMAN, MT 59715
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (19 JUL 1996) Vol. 271, No. 29, pp. 17013-17020.
ISSN: 0021-9258.
PUBLISHER: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 55
ENTRY DATE: Entered STN: 1996

Last Updated on STN: 1996
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The human neutrophil ***NADPH*** ***oxidase*** is a multi-component complex composed of membrane-bound and cytosolic proteins, During activation, cytosolic proteins p47(phox) P67(phox), Rac2, and possibly p40(phox) translocate to the plasma membrane and associate with flavocytochrome 5 to form the active superoxide-generating system, To further investigate the role of p67(phox), this complex assembly ***process*** , experiments were performed to ***identify*** possible regions of interaction between p67(phox) and other ***NADPH*** ***oxidase*** proteins, Using random ***sequence*** peptide phage display library analysis of p67(phox) we ***identified*** a novel region in p47(phox) encompassing residues 323-332 and a previously ***identified*** SH3 binding domain encompassing p47(phox) residues 361-370 as p67(phox) binding sites, Synthetic peptides mimicking p47(phox) residues 323-332 ***inhibited*** the p47(phox)-p67(phox) binding interaction in an affinity binding assay; however, peptides mimicking flanking regions were inactive, Surprisingly, this same region of p47(phox) was found previously to represent a site of binding interaction for flavocytochrome b (DeLeo, F, R,, Nauseef, W, M,, Jesaitis, A. J,, Burritt, J, B,, Clark, R, A., and Quinn, M. T, (1995) J, Biol. Chem, 270, 26246-26251), and this observation was confirmed in the present report using two different in vitro assays that were not evaluated previously, Using affinity binding assays, we also found that p67(phox) and flavocytochrome 5 competed for binding to p47(phox) after activation, suggesting that prior to full NADPH oxidase assembly the 323-332 region of p47(phox) is associated with p67(phox) and at some point in the activation process is transferred to flavocytochrome b, Thus, taken together our data demonstrate that both p67(phox) and flavocytochrome b utilize a common binding site in p47(phox) presumably at distinct stages during the activation process, and this p47(phox) region plays a key role in regulating NADPH oxidase assembly.

L10 ANSWER 57 OF 62 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.
on STN DUPLICATE 16
ACCESSION NUMBER: 1996-0143923 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1996 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Analysis of the priming activity of lipids generated during routine storage of platelet concentrates
AUTHOR: , SILLIMAN C. C.; DICKEY W. O.; PATERSON A. J.; THURMAN G. W.; CLAY K. L.; JOHNSON C. A.; AMBRUSO D. R.
CORPORATE SOURCE: Univ. Colorado school medicine, bonfils memorial blood cent., dep. pediatrics, Denver CO 80262, United States
SOURCE: Transfusion : (Philadelphia, PA), (1996), 36(2), 133-139, 43 refs.
ISSN: 0041-1132 CODEN: TRANAT
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-10224, 354000053031290090
AN 1996-0143923 PASCAL
CP Copyright .COPYRGT. 1996 INIST-CNRS. All rights reserved.
AB Background : ***Compounds*** generated during the routine storage of platelet concentrates may have deleterious effects on the transfusion recipient. Study Design and ***Methods*** : Daily plasma samples from platelet concentrates, both apheresis platelets and those separated from whole blood, were obtained serially during routine storage. These plasma samples were assayed for their ability to prime the ***NADPH*** ***oxidase*** in ***isolated*** human neutrophils. Quantitative and qualitative analysis of the priming agents was completed by lipid extraction, high-pressure liquid chromatography separation, and gas chromatography/mass spectroscopy. Results : ***Compounds*** were generated in both apheresis and whole-blood platelets that significantly primed the ***NADPH*** ***oxidase*** after 24 and 48 hours of storage, respectively. The priming activity was maximal by component outdate : 2.6-fold that of the buffer-treated control neutrophils (apheresis) and 3.9-fold that of the buffer-treated control neutrophils (whole blood). These agents were generated by cellular constituents, as

stored plasma did not demonstrate such priming activity. ***Inhibition*** of this priming activity by WEB 2170, a specific platelet-activating factor receptor ***antagonist*** , suggested that the observed priming involved the platelet-activating factor receptor. A portion of the priming activity from platelet concentrates was organically extractable : 69 percent of that from apheresis platelets and 46 percent of that from whole-blood platelets. Further purification of the lipid's priming activity by normal-phase high-pressure liquid chromatography demonstrated a single peak of priming activity at the retention time of lysophosphatidylcholines. Because 46 percent of the priming activity from whole-blood platelets was chloroform insoluble and because it has been reported that interleukin 8 is generated during routine storage of whole-blood platelets, the effects of interleukin 8 on the ***NADPH*** ***oxidase*** were examined. ***Recombinant*** monocyte interleukin 8 rapidly primed the ***oxidase*** but was not ***inhibited*** by WEB 2170. Conclusion : Lipids were generated during the routine storage of platelet concentrates that prime the ***NADPH*** ***oxidase*** , and they may play a role in the severe complications of transfusion therapy. Other non-lipid ***compounds*** , such as interleukin 8, that are generated in whole-blood platelets may also contribute to the observed priming activity of plasma.

L10 ANSWER 58 OF 62 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 1995:25365747 BIOTECHNO
TITLE: 'Peptide walking' is a novel method for mapping functional domains in proteins. Its application to the Rac1-dependent activation of NADPH oxidase
AUTHOR: Joseph G.; Pick E.
CORPORATE SOURCE: Julius Friedrich Cohnheim CPR, Sackler School of Medicine, Tel Aviv University,Tel Aviv 69978, Israel.
SOURCE: Journal of Biological Chemistry, (1995), 270/49 (29079-29082)
CODEN: JBCHA3 ISSN: 0021-9258
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1995:25365747 BIOTECHNO
AB Activation of the superoxide generating ***NADPH*** ***oxidase*** of phagocytes involves the assembly of a multimolecular complex and is dependent on the participation of the small molecular weight GTP-binding protein Rac (1 or 2). This model system was used for mapping functional domains in the primary ***sequence*** of Rac1, based on assessing the ***inhibitory*** effect of 90 individual overlapping pentadecapeptides, spanning the entire length of Rac1, on ***NADPH*** ***oxidase*** activation in two types of cell-free assay. Five functional domains were ***identified*** , each consisting of a cluster of contiguous residues shared by members of five groups of overlapping ***inhibitory*** peptides. Four of the five domains are exposed on the molecular surface of Rac1 and were not ***identified*** previously by mutational analysis; the fifth corresponds to a polybasic motif near the carboxyl terminus, confirming earlier reports. ***Screening*** the entire linear ***sequence*** of a protein with a battery of overlapping peptides for interference with its ability to interact with upstream or downstream molecules should be of wide applicability as a reliable, fast, and economical ***method*** for mapping of functionally relevant domains.

L10 ANSWER 59 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 1996008568 ESBIOBASE
TITLE: Inhibition of human glutathione reductase by S-nitrosoglutathione
AUTHOR: Becker K.; Gui M.; Schirmer R.H.
CORPORATE SOURCE: K. Becker, Institute of Biochemistry II, IFN 328, D-69120 Heidelberg, Germany.
SOURCE: European Journal of Biochemistry, (1995), 234/2 (472-478)
CODEN: EJBCAI ISSN: 0014-2956

DOCUMENT TYPE: Journal; Article
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
SUMMARY LANGUAGE: English
AB S-Nitrosoglutathione (GSNO) represents a major transport form of nitric oxide (NO) in biological systems. Since NO and GSNO have been shown to modulate the function of various proteins, we studied the influence of GSNO and other NO donors on human glutathione reductase (GR). Catalyzing the reaction ***NADPH*** + GSSG + H.sup.+ .fwdarw. NADP.sup.+ + 2 GSH, the dimeric flavoprotein GR is the central enzyme of the glutathione redox metabolism. GSNO was found to ***inhibit*** crystalline erythrocyte GR in two ways: (a) as a reversible ***inhibitor*** GSNO is competitive with glutathione disulfide (GSSG), the K(i) being appr. 0.5 mM; (b) as an irreversible ***inhibitor*** ; after 1 h (3 h) incubation with 1 mM GSNO, GR (2.5 U/ml, representing intraerythrocytic concentrations) was ***inhibited*** by 70% (90%). This ***inhibition*** depended on the presence of ***NADPH*** and could not be reversed by dilution nor by reducing agents. Absorption spectra indicate that the charge transfer interaction between Cys63 and the flavin is abolished by this modification. In a GR sample ***inhibited*** by 90% with GSNO, the K(m) values for the substrates GSSG and ***NADPH*** were not significantly changed nor did the modification induce ***oxidase*** activity of the enzyme. GSNO was found not to be a substrate in the forward reaction of GR. This implies that GSNO is not accounted for by ***methods*** which employ GR for ***determining*** total glutathione. Incubating ***isolated*** GR for 60 min with other NO donors, namely 1 mM sodium nitroprusside or 1 mM S-nitroso-N-acetyl-DL-penicillamine (SNAP), resulted in only 25% and 10% ***inhibition*** , respectively. This attests to a specific affinity of GSNO to the enzyme. GSNO ***inhibition*** patterns comparable to purified authentic GR were obtained for purified ***recombinant*** GR, a GR mutant lacking the 15 N-terminal amino acids including Cys2, and for the enzyme present in diluted fresh haemolysates (0.02 U/ml); in concentrated haemolysates the ***inhibition*** was less pronounced. GR of intact erythrocytes was not affected when exposed to GSNO in the medium. Our results suggest that the irreversible ***inhibition*** of GR by GSNO involves nitrosylation of Cys63 and/or Cys58 at the catalytic site of the enzyme. To further investigate the mechanism of inactivation we have crystallized GSNO-modified GR for X-ray diffraction analysis.

L10 ANSWER 60 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 1995150921 ESBIOBASE
TITLE: Characterization of neutrophil NADPH oxidase activity reconstituted in a cell-free assay using specific monoclonal antibodies raised against cytochrome b.sub.5.sub.5.sub.8
AUTHOR: Batot G.; Martel C.; Capdeville N.; Wientjes F.; Morel F.
CORPORATE SOURCE: F. Morel, Laboratoire d'Enzymologie, CHU, F-38043 Grenoble Cedex 9, France.
SOURCE: European Journal of Biochemistry, (1995), 234/1 (208-215)
CODEN: EJBCAI ISSN: 0014-2956
DOCUMENT TYPE: Journal; Article
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The immunochemical characterization of ***NADPH*** ***oxidase*** activity of cytochrome b.sub.5.sub.5.sub.8 purified from human neutrophils was ***determined*** after reconstitution in a cell-free assay using the native hemoprotein and ***recombinant*** purified cytosolic activating factors. The ***oxidase*** activity showed a strict dependence on the heme content at each step of the hemoprotein purification ***process*** . The immunochemical properties of the reconstituted ***oxidase*** made use of monoclonal antibodies raised against membrane-bound and octyl-glucoside-extracted cytochrome b. From nine specific monoclonal antibodies reacting with gp91-phox cytochrome b.sub.5.sub.5.sub.8, two were selected, both of which were found to bind to the .beta. subunit of cytochrome b.sub.5.sub.5.sub.8 and to

inhibit superoxide formation in the ***oxidase*** reconstituted cell-free assay. The extent of ***inhibition*** was dependent on the phospholipid environment. Neutrophil membrane extracts from X-linked chronic granulomatous disease patients did not produce O.sub.2.sup.- in the reconstituted system and did not bind to the antibodies.

L10 ANSWER 61 OF 62 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 1994095115 ESBIOBASE
TITLE: An SH3 domain and proline-rich sequence mediate an interaction between two components of the phagocyte NADPH oxidase complex
AUTHOR: Finan P.; Shimizu Y.; Gout I.; Hsuan J.; Truong O.; Butcher C.; Bennett P.; Waterfield M.D.; Kellie S.
CORPORATE SOURCE: P. Finan, Yamanouchi Research Institute, Littlemore Hospital, Oxford OX4 4XN, United Kingdom.
SOURCE: Journal of Biological Chemistry, (1994), 269/19 (13752-13755)
CODEN: JBCHA3 ISSN: 0021-9258
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Neutrophils possess a multicomponent ***NADPH*** ***oxidase*** system capable of producing large quantities of superoxide in a ***process*** known as the respiratory burst (1). Upon stimulation of a phagocytic cell, two cytosolic components of the ***oxidase*** , p67(phox) and p47(phox), associate with a membrane-bound flavocytochrome b and a small GTP-binding protein to form a functional enzyme complex. Each of the Phox proteins contains two src homology 3 (SH3) domains, which are of unknown function but are potential mediators of protein-protein interactions between components of the activated ***oxidase*** . We have ***isolated*** a 47-kDa protein from lysates of differentiated HL60 cells that specifically bound to the carboxyl-terminal SH3 domain of p67(phox) and not to any other SH3 domain tested. This protein was ***identified*** as p47(phox), and the putative SH3 domain binding site was located to a carboxyl-terminal proline-rich region. Proline-rich synthetic peptides based on this carboxyl-terminal region specifically ***inhibited*** the binding of p47(phox) to the carboxyl-terminal SH3 domain of p67(phox), and sequential truncation defined a unique minimal ***sequence*** , which, although similar, does not match the consensus ***sequence*** defined for other SH3-binding proteins.

L10 ANSWER 62 OF 62 CANCERLIT on STN
ACCESSION NUMBER: 95607328 CANCERLIT
DOCUMENT NUMBER: 95607328
TITLE: The role of specific reductases in the intracellular activation and binding of 2-nitroimidazoles (Meeting abstract).
AUTHOR: Chapman J D; Stobbe C C; Joseph P; Jaiswal A K
CORPORATE SOURCE: Department of Radiation Oncology, Fox Chase Cancer Center, 7701 Burholme Avenue, Philadelphia, PA 19111.
SOURCE: Non-serial, (1993) Eighth International Conference on Chemical Modifiers of Cancer Treatment, June 16-19, 1993, Kyoto, Japan, p. 89-90, 1993. .
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950405
Last Updated on STN: 19950405
AB Nitroimidazole (NI) ***compounds*** are reduced within mammalian cells to activated intermediates which can covalently bind with cellular molecules. These reactions are enzyme mediated and dependent upon NI structure and concentration, oxygen concentrations, cell type and temperature. Hydroxylamine- and nitroso-intermediates had been shown to efficiently bind to cellular molecules. This study was performed to ***determine*** the relative importance of xanthine ***oxidase*** ,

DT-diaphorase and P450 reductase in activating NI within cells. Both enzyme ***inhibition*** and plasmid transfection studies were performed with monkey kidney cells, and covalent binding of 14C-NI to cellular macromolecules was measured. Binding rates (BR) of NI were assumed to be indicative of steady-state intracellular concentrations of activated intermediate. Materials and ***methods*** : COS 1 (monkey kidney) cells were grown as monolayers and transfected with the ***recombinant*** plasmids, pMT2- ***NADPH*** : cytochrome P450 oxidoreductase and pMT2-DT diaphorase. P450 reductase and DT-diaphorase activities in cell extracts were quantified by enzyme assays. Concentrations of allopurinol known to ***inhibit*** the activity of intracellular xanthine ***oxidase*** had little or no effect on the binding rates of NI to hypoxic COS 1 cells. This result is indirect evidence that xanthine ***oxidase*** is relatively unimportant for the intracellular reduction of these NIs to activated intermediates which can bind to cellular molecules. A 1000 x overexpression of DT-diaphorase in COS 1 cells by plasmid transfection resulted in a 1.3 x increase in hypoxic BR of NI. This result suggests that DT-diaphorase plays a relatively minor role in the reduction of NI to activated intermediates which can bind to cellular molecules. An 80 x overexpression of P450-reductase in COS 1 cells resulted in a 5-8 x increase in NI BR. This result suggests that P450 reductase is the most important cellular enzyme of the three investigated for reducing NI to intermediates which can bind to cellular molecules. The approx 7 x increase in BR which results from the 80 x increase in intracellular enzyme activity is consistent with 1/2-order kinetics. Our previous studies on hypoxic binding kinetics of NI to EMT-6 and V-79 cells had shown that a 10 x increase in binding rate resulted from a 100 x increase in concentration of NI substrate. One-half order kinetics of cellular binding of misonidazole and desmethylmisonidazole to cellular macromolecules in mammalian cells can, consequently, be demonstrated by modulating either the enzyme or the substrate concentrations.

=> d his

L1 QUE (NADPH(S) OXIDASE#) OR (DUAL(S) OXIDASE#) OR NOX1 OR NOH1

FILE 'BIOSIS, SCISEARCH, CAPLUS, TOXCENTER, MEDLINE, EMBASE, ESBIOBASE, PASCAL, BIOTECHNO, LIFESCI, USPATFULL, CANCERLIT' ENTERED AT 10:04:59 ON 24 NOV 2005

L2 43932 S L1
L3 4878 S (SCREEN? OR ISOLAT? OR FIND? OR DETERM?) (S) L2
L4 6284 S (SCREEN? OR ISOLAT? OR FIND? OR DETERM? OR IDENTIF?) (S) L2
L5 2210 S (SUBSTANC? OR COMPOUND? OR INHIBIT? OR ANTAGONI?) (S) L4
L6 424 S (METHOD? OR PROCESS?) (S) L5
L7 93 S (GENE# OR SEQUENCE# OR CLONE# OR POLYNUCLEOTIDE# OR RECOMBINA
L8 16 S DISEASE# (S) L7
L9 1 S RHEUMATOID (S) L7
L10 62 DUP REM L7 (31 DUPLICATES REMOVED)

=> log y